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THE ELECTRICAL RESPONSE OF THE EYE TO LIGHT

BY
SIR JOHN PARSONS, F.R.S.

THE researches of Keith Lucas (1909-1914), extended by Adrian, have shown that every nerve impulse in the body is accompanied by an electrical response. After a short latent period the stimulus sets up a train of changes in potential following each other in rapid succession, varying from 10 to 300 per second. Each response is followed by a short refractory period of 2-3 σ $\left(\sigma = \frac{1}{1000}\text{th. sec.}\right)$ during which no stimulus, however strong, produces any response. The response is of the "all-or-none" type, *i.e.*, if it is above the threshold it produces the same change in potential. Increase in strength of the stimulus merely increases the frequency of the waves of electrical change. The normal, so-called "adequate," stimulus of any sensory end organ produces exactly the same type of discharge *in the nerve* as stimulation of the nerve by artificial means, *e.g.*, an electric shock. These comparatively simple laws are found to hold good for all nerves, both efferent and afferent. The changes are so rapid that abolition of inertia in the recording apparatus is of the greatest importance. The replacement of the capillary electrometer, used in the early experiments, by the string galvanometer was a great advance, further improved by the enormous magnification which thermionic valve amplifiers provide.

If non-polarizable electrodes are placed on the cornea and optic nerve of an animal and are joined by a wire, with a galvanometer in the circuit, it is found that a current flows from the cornea through the galvanometer towards the optic nerve (du Bois-Reymond, 1849). This, the *current of rest,* has a difference of potential of 7 to 9 millivolts, and is probably the integration of several changes of potential, some of which are purely physical, others physiological. The Donnan difference of potential between blood and aqueous is probably one purely physical factor. It is found, however, that, so far as the retina is concerned, the retinal factor is always of such a nature that the neuroepithelial layer is negative to the nerve-fibre layer; so that the direction of the

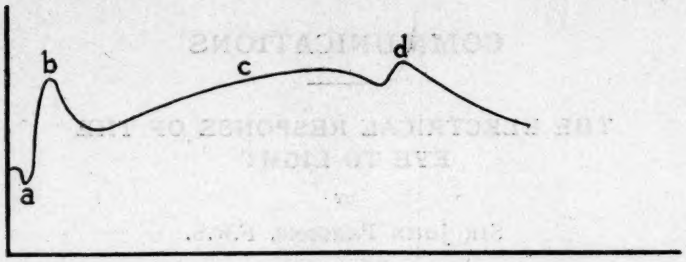

FIG. 1.

Typical Electroretinogram.

current outside the retina is in opposite directions in vertebrates and invertebrates (Brücke and Garten, 1907; Riedel, 1918), since the neuroepithelium is directed away from and towards the anterior part of the eye respectively in these groups.

If the current of rest is compensated for and light is thrown upon the eye a *current of action* is induced (Holmgren, 1865), but only so long as the eye survives: it is abolished by removal of the retina from the eye (Dewar and M'Kendrick, 1873), and by anaesthetics. The first accurate measurements were made by Gotch (1903-4) with the capillary electrometer. He showed that in the frog monochromatic red light gave a latent period of 0·3 sec. and a difference of potential of 0·4 millivolt; green 0·2 sec. and 0·5 m v; violet 0·25 sec. and 0·24 m v. The researches of many observers, especially those of Piper (1904-11), Einthoven and Jolly (1909), Kohlrausch (1913-25), Adrian and Matthews (1927) and Granit and his co-workers (1933), have shown that the typical response is as follows (Fig. 1). (1) A latent period; (2) a very brief negative phase (*a*); (3) a rapid steep initial positive phase (*b*): (4) a slow depression; (5) a slow secondary positive

phase (*c*), persisting for a long time under continued stimulation. On removal of the light there is (6) a short latent period; (7) a sharp positive phase (*d*) gradually diminishing to the resting state.

The general trend of the responses and especially the unexpected negative phase (*a*) and the strong positive phase (*d*) on removal of the light, indicate that the reaction is of a composite character. This is not surprising in view of the fact that at least three, and probably far more, neurones are involved. It cannot be doubted that complete analysis of the response would throw a flood of light on the physiology of vision, and the preliminary steps, taken particularly by Piper, Einthoven and Jolly, Adrian, and Granit, confirm this expectation.

In the first place, there is no doubt that the current originates in the retina, for it is abolished if the retina is removed (Dewar and M'Kendrick, 1873), but remains unchanged if the anterior part of the eye is removed (Bowie, Chaffee and Hampson, 1923). The curve is much simpler in cephalopods (Beck, 1899; Piper, 1911; Fröhlich, 1913), probably owing to the greater simplicity of the retina and the aggregation of the ganglion cells in a mass posterior to the eyeball. Arthropod eyes show transitions towards the mammalian type of curve (Hartline, 1927). The *a* and *d* responses are more marked in fishes, amphibia, reptiles, and birds than in mammals. In extirpated frog's eyes the difference of potential may reach 2·8 m v, but is usually less than 1 m v in warm-blooded animals.

The difference of potential varies with the position of electrodes (de Haas, 1903; Westerlund, 1912), being greatest between the anterior and posterior poles (Westerlund). Loss of vitality (Kühne and Steiner, 1880) injury massage (*e.g.*, Waller, 1903, 1909; Jolly, 1909), cold (Nikiforowsky, 1911), some drugs, *e.g.*, K, Ba, in less degree Mg, and Ca salts added to the Ringer's solution (Beuchelt, 1921), tend to eliminate the *b* response and emphasize the *a* and *d* responses. With temperature and drugs the reactions are reversible. Asphyxia eliminates the *b* response, but the *c* response is retained if the eye is dark-adapted (Kohlrausch, 1918); this reaction is also reversible. With progressive narcotization in the decerebrate cat the *c* response disappears first; the other responses are diminished (*vide infra*).

Adaptation. The sensitivity of the retina to light is enormously increased by prolonged (½ hr. or more) exclusion of light from the eye. When in a condition of dark-adaptation vision is said to be scotopic, in light-adaptation photopic.

The effect on the electroretinogram of change from dark- to light-adaptation differs in different animals. Generally speaking the reaction is greater in scotopia (Kühne and Steiner, 1880; Brücke & Garten, 1907). As the eye becomes light-adapted the

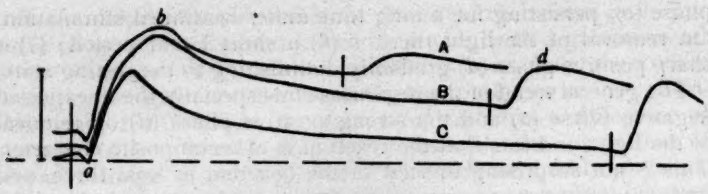

FIG. 2.

Frog. A, dark adapted; B, 3 minutes after A (the eye being in the dark meanwhile); C, light adapted. Vertical line = 0·673 mv.; short vertical lines show on and off of stimuli—1 second in A; 1·5 second in B; 2 seconds in C. (Granit and Riddell, 1934.)

latent period is increased, the *a* response is generally increased, the *b* wave is diminished and rises less steeply, the *c* wave is abolished, and the *d* wave enhanced (*cf.* Fig. 10).

An almost pure *b* curve can be obtained with the frog's eye by good dark-adaptation and a stimulus of 0·5-1 sec. with very weak light (Einthoven and Jolly, 1909). With a strong stimulus applied to the photopic eye the *a* and *d* waves are always manifest, and under all circumstances these vary together.

Variations of great theoretical interest occur in different animals. These appear to be related to their evolutionary status, retinal structure, and habits of life. It is impossible at present to correlate the electrical reactions with retinal structure, but it cannot be doubted that these reactions, however apparently contradictory at present, will throw much light on the problem.

It has been found by Kohlrausch (1918) that in pigeons and fowls, which are essentially diurnal birds, the *c* wave is present only in the condition of light-adaptation, and diminishes with dark-adaptation, the reaction being reversible. Other diurnal animals, such as the turtle (Emys europæa), buzzard, and probably the dog behave similarly; whereas nocturnal animals, such as the pike, salamander, owl, and rabbit behave like the frog.

It has already been pointed out that the secondary positive *c* response is abolished by light-adaptation in amphibian and mammalian eyes. The other phases are more difficult to eliminate. Waller (1909) showed that after massage it was possible to obtain a purely negative response in the frog—an initial fall with the latent period of the *a* wave and a quick rise to the base line on cessation of stimulation. Granit and Riddell (1934) confirmed this observation except that they failed to obtain complete cessation of the *b* response. It has been shown by Nikiforowsky (1912) and Tirala (1917) that the *b* and *d* responses may vary independently. During light-adaptation the *b* wave remains practically constant; adaptation is essentially associated with the *a* wave.

Narcotization affords another means of analysis of the curve. Working on the dark-adapted eye of the decerebrate cat Granit (1933) used two intensities of stimulation, viz., 14 millilamberts and 0·14 ml. The *a* and *d* waves are present only with high intensities or large areas of stimulation. Diminution of area stimulated acts like diminution of intensity, showing abolition of the *a*, *c*, and *d* waves. The *c* wave is eliminated by small doses of ether, leaving the more rapid *a* and *b* waves unaltered, the *d* effect being enhanced. The *c* wave itself, however, is composite, for though the major part is abolished by slight anaesthesia,

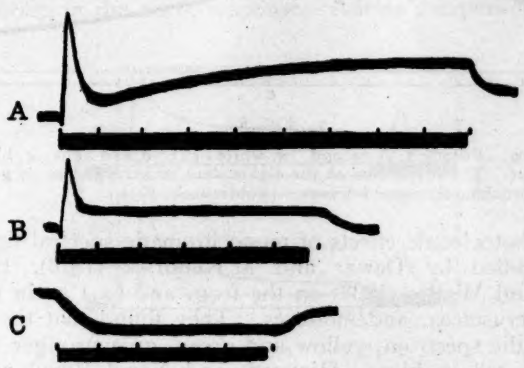

FIG. 3.

Decerebrate cat. Progressive effect of anaesthesia with stimulus of 1·4 millilambert. A, before narcotization; B, after 21 minutes of anaesthesia; C, after 31 minutes *b*-wave in A = 0·417 mv. (Granit, 1933.)

deeper anaesthesia is necessary to abolish the remainder. At high intensity of stimulus ether abolishes 50 per cent. of the rise, whereas at low intensities only about 8 per cent. diminution occurs. Further narcotization slows and diminishes the *b* wave and lengthens its latent period. Similarly the negative *a* response becomes slower with longer latent period. In the final stage only the negative component persists (Fig. 3).

Wave-length. It has already been mentioned that Gotch (1903-4) measured the electrical reactions of the frog's eye to prolonged stimulation with white and monochromatic red, green and violet lights. The reactions of the dark-adapted frog's eye to momentary (1/15 sec.) stimulation with white, red, and blue lights are shown in Fig. 4 (Kohlrausch, 1925). The filters were so arranged that the *b* waves were practically equal; it will be noted that the *c* wave is negligible with red, and much enhanced with blue light.

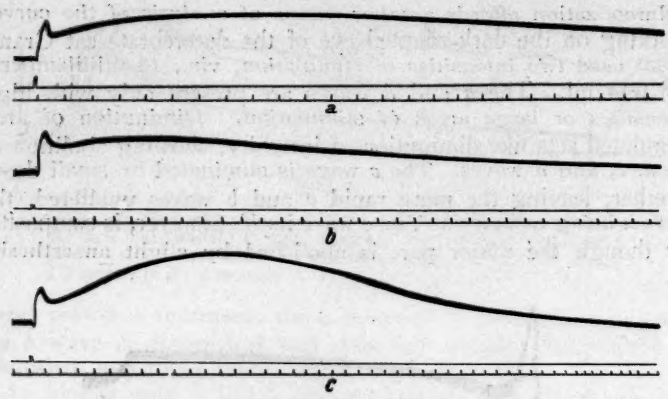

FIG. 4.

Frog. Stimulus, $\frac{1}{10}$ second. *a*, white light; *b*, red light; *c*, blue light. The intensities of the lights were so arranged as to give approximately equal *b*-waves. (Kohlrausch, 1925.)

The photoelectric effects of monochromatic spectral lights have been studied by Dewar and M'Kendrick (1876), Holmgren (1880), and Waller (1903) on the frog, and by Chatin (1880) on insects, crustacea, and molluscs. They found that the brighter parts of the spectrum, yellow and green, give stronger responses than the red or blue. Himstedt and Nagel (1900) and Piper (1904-5) made comparative estimates throughout the spectrum. Himstedt and Nagel found that the maximum with fairly strong stimuli for the light-adapted frog was at about 586$\mu\mu$; feeble stimuli for the dark-adapted frog about 550$\mu\mu$. The photoelectric response curves resemble nearly the photopic and scotopic luminosity curves of man. The shift of the maximum is the same in each case, thus proving that the Purkinje phenomenon manifests itself already in the retina and is therefore not fundamentally dependent upon central nervous processes. Piper showed that diurnal birds (pigeon, fowl, buzzard) gave the photopic reaction; nocturnal birds (owls) the scotopic reaction, as did also dogs, cats and rabbits irrespective of the condition of adaptation. There is a still further striking shift of the curve in the cephalopod eye (Piper).

Kohlrausch and Brossa (1914) and Kohlrausch (1918) found that with diminishing wave-length the *a* and *c* waves become enhanced, markedly in the photopic frog and diurnal birds, very slightly in the scotopic frog and nocturnal birds. Kahn and Löwenstein (1924), Hartline (1925), and Sachs (1929) have demonstrated the same fact for man.

It is interesting to note that Kohlrausch (1918) has shown that the response with purple light in the pigeon is an exact interference curve due to summation of the red and blue responses.

The Optic nerve Response. Before discussing the more complex problem of the effects of variations in intensity, area, and time of exposure of the stimulus upon the electroretinogram it will be well to review our knowledge of the simpler problem of the response in the optic nerve—knowledge which we owe chiefly to Adrian.

Ishihara (1906) and Westerlund (1912) had already shown that the typical positive "on" and "off" effects of the retinal response also occurred in the nerve response. Adrian's experiments were

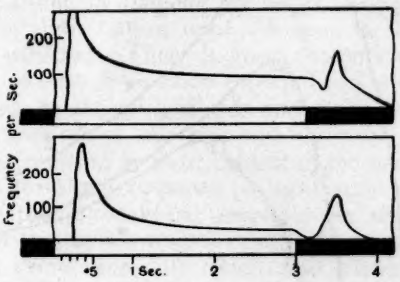

FIG. 5.

Conger eel. Frequency of impulses per second during and after stimulation. Illumination = 830 metre candles; disc diameters = 36 and 12·8 mm. (Adrian and Matthews, 1927.)

made chiefly upon the optic nerve of the conger eel, which is specially long and well suited for the purpose. In many of the experiments only the posterior part of the eye was retained, the retina was stimulated with light, and both electrodes were applied to the nerve, with an interval between them. Adrian and Matthews (1927-8), as already mentioned, showed that the responses in the optic nerve agreed in all respects with the responses in other nerves. Changes in intensity of the stimulus therefore do not cause changes in intensity of the reaction, but manifest themselves as changes in the *frequency* of periodic oscillations.

Fig. 5 shows the typical response of a dark-adapted eye to steady (3 secs.) illumination and the effect of altering the area of retina illuminated. There is a latent period of about 0·2 sec., followed by a sudden outburst of very rapid oscillations. The oscillations diminish in frequency, rapidly at first and then more slowly, whilst the stimulus is still acting. Cessation of the excitation causes a small fall in frequency, followed by a well-marked

rise, after which the frequency rapidly diminishes to zero. Clearly we have here the optic nerve expression of the *b* and *d* waves of the electroretinogram. There is no manifest evidence of the *a* and *c* curves. Increase of the area or intensity of the stimulus causes shortening of the latent period and increased frequency of the oscillations.

The fall of the curve during the continuance of steady illumination is evidence of adaptation, and may be directly correlated

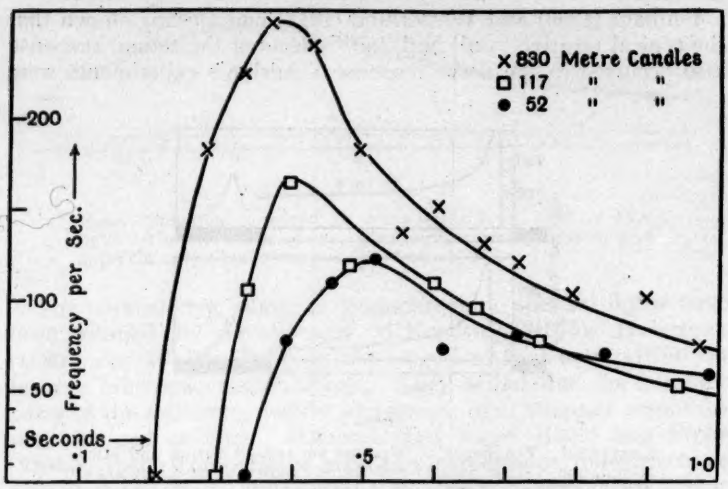

FIG. 6.

Conger eel. Evolution of discharge with different intensities of illumination. Disc diameter = 36 mm. (Adrian and Matthews, 1927.)

with visual sensation; for it is well known that prolonged fixation of an illuminated surface leads to a diminution of the subjective brightness. If during exposure, a shadow is moved across the visual field, the discharge during the movement is far greater than during continuous exposure. This is already correlated with the great sensitivity of the eyes of lower animals and of the peripheral field in man to movement of objects within the field of vision.

A short stimulus (*e.g.*, 0·125 sec.) causes a discharge of similar form lasting about 1 sec. after a latent period of about 0·2 sec. from the beginning of the stimulus. The fall of the curve is more rapid and there is no "off" effect. Feeble illumination or small area stimulated will give a similar curve with a continuous illumination, but with stronger lights and larger areas there is always

a definite "off" effect provided that the stimulus has lasted for a second or more.

Increase of intensity of the stimulus causes reduced latent period, increased maximum frequency of the discharge and more rapid rise of the frequency curve (Fig. 6). The maximum frequency curves show that the frequency is some exponential function of the intensity (*cf.* Weber-Fechner Law).

Increase of area stimulated, with constant intensity, has exactly similar effects. Since the maximum frequency is not directly proportional to the area stimulated and the latent period is reduced when the area is increased, it follows that the retina is not a mosaic of isolated points, but that there is interaction between the points stimulated. This interaction is of such a nature that for areas of less than about 1 mm. in diameter the response depends upon the total quantity of light falling upon the area in unit time, *i.e.*, $i \times a \times t$ = a constant. There is good anatomical evidence for the probability of such interaction especially in the periphery of the human retina, in the ratio of rods and cones to one ganglion cell and in the presence of amacrine and horizontal cells, but one would expect the result to be most evident at the periphery. If we can regard the threshold response to light stimulus as directly correlated with the photoelectric response we should expect a similar result in the human retina. Curiously enough the law is valid for foveal stimulation only (Riccò and others).

Analysis of the Electroretinogram. Piper (1911), confirmed by Kohlrausch (1918), found that vertebrates (fish, amphibians, and reptiles) which had large *a* waves also had good off-effects (*d*), whereas those (*e.g.*, rabbit, cat and dog) which gave small and inconstant *a* waves also had similar *d* waves. They therefore ascribed the *a* and *d* waves to the same process. The same conclusion has been reached by Einthoven and Jolly (1908), and is further confirmed by Adrian and Matthews (1927).

Adrian and Matthews proved that the discharge in the optic nerve commences at a fixed time (about 0·1 sec.) after the beginning of the retinal response, irrespective of the length of the latent period of the latter. This constant delay must be due to changes in the retina, and may be due either to photo-chemical changes or to delay in the two sets of retinal synapses through which the impulse must pass. They further showed that the retinal nerve intervals for *a* and *d* were the same, and that therefore both must be of the same nature.

There can be no doubt that the electrical response is of a composite nature (Kühne and Steiner, 1880), and various attempts have been made to analyse the curve into its fundamental constituents. These attempts are highly theoretical, and must be regarded as suggestions on which to base further research rather

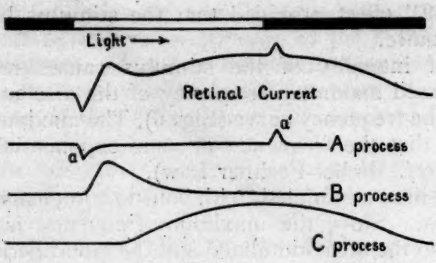

FIG. 7.

Einthoven's analysis of the electroretinogram.

than representations of proved facts. Einthoven's analysis is shown in Fig. 7. The component A is responsible for the *a* and *d* waves. The component B is responsible for the *b* wave, which is most marked with feeble illumination. The slow *c* wave, often persisting beyond the end of excitation, is subserved by the C component. The least satisfactory of these components is A, for Waller's experiment, as well as many other observations since, have emphatically indicated that the A process is entirely negative, in spite of the positive off-effect (*d*).

Piper's analysis is shown in Fig. 8. His component II corresponds to Einthoven's A, and is seen to be purely negative. I corresponds with B, and II with C.

The most recent and exhaustive attempt is that of Granit, which is a modification of Piper's. He has adduced evidence to show

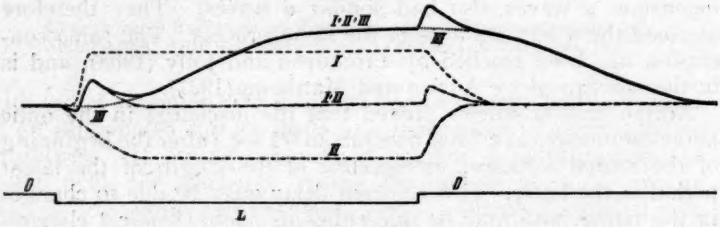

FIG. 8.

Piper's analysis of the electroretinogram.

that *c* is itself composite, and that a large proportion of it can be easily eliminated with ether. This part he calls PI. The positive component of the total curve remaining after the elimination of PI reacts uniformly and simultaneously to ether at all intensities of stimulation, and is PII. The negative component is PIII. These are combined as shown in Fig. 9.

The response to a weak stimulus gives an almost pure PII. "By using ether it was possible to obtain PII alone at low intensities, PIII alone at high intensities, and also to produce the response PII + PIII. By interfering with the oxygen supply it was possible to produce the response PI + PIII. It has not been possible to obtain PI alone. Thus it follows that PII and PIII are very directly related to the stimulus. It does not seem probable that the one elicits the other. As to PI, it may, of course, merely be a matter of finding the right procedure in order to obtain it alone, but so far it seems as if PI required PIII. It does not

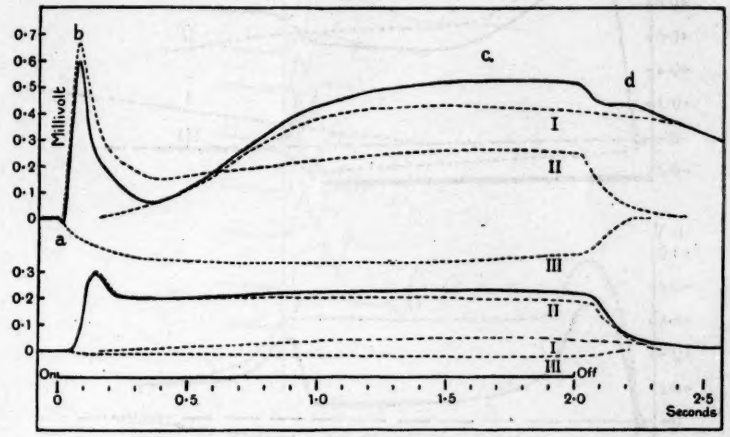

FIG. 9.

Granit's analyses of the electroretinogram with different intensities of stimulation—14 ml and 0·14 ml. and area of 1661 sq. mm. at a distance of 70 mm. (Granit, 1933.)

require PII to judge from the large PI in the response PI + PIII."

The ether experiments often show an enhanced off-effect after removal of PI. On the other hand, removal of PII at high intensities regularly increases the off-effect (Kohlrausch, Granit). Hence, the component necessary for the relatively positive *d* wave to appear is the negative PIII plus either PI or PII. For the alternative explanation that PIII actually rises above the base line at the end of stimulation (*cf.* Einthoven) there is no evidence. Since PI and PII drop at the cessation of the stimulus, it follows that the absence of either counter-acting fall must make the off-effect caused by the return to zero of PIII more marked. PII drops faster than PI; therefore the removal of PII must cause particularly large off-effects, which actually is the case.

The schematic analyses of the responses of the frog's eye in dark- and light-adaptation are shown in Fig. 10.

Intermittent Light. The response to stimulation with intermittent light has been studied by Kühne and Steiner (1880), Piper (1911), Day (1915), Creed and Granit (1933), and Granit and Riddell (1934). Piper found that the response consists of

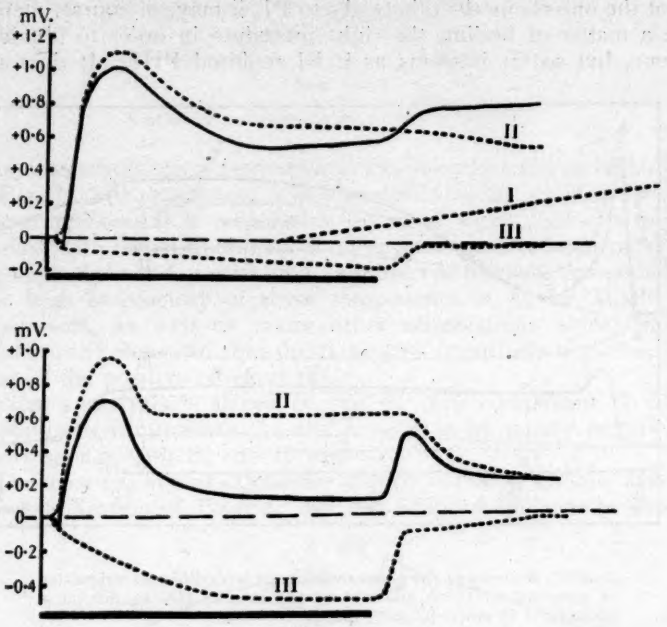

FIG. 10.

Granit's analyses of the electroretinogram of the frog in dark adaptation (above) and in light adaptation (below). Duration of stimulus (thick horizontal line) = 2 seconds. (Granit and Riddell, 1934.)

undulations during the slow phase (*i.e.*, on the *c* wave), which disappear above a certain frequency. This critical frequency is low in eyes containing mainly rods (cat), and much higher in eyes in which cones predominate (pigeon). Renqvist (1924) noted that flashes superposed upon steady illumination of the frog's eye gave rise to small *a* and *b* waves superposed upon the slow *c* wave. Adrian and Matthews (1928) recorded the impulses in the optic nerve of the conger eel, and found that below a certain frequency, increasing with the area or intensity of the stimuli, the discharge waxed and waned at the same rhythm as the stimulation, but that

above that frequency there was a steady stream of impulses such as is given with continuous illumination.

Creed and Granit (1933), working with the decerebrate cat, confirmed Piper's results. With intense stimuli and a well-developed *b* wave, the undulations only became visible on the fall of the *b* wave, and are smaller during the first second than afterwards. With weaker stimuli and a smaller *b* wave the ripples corresponding to the rate of stimulation appear at the beginning and may be actually larger and steeper than the *b* wave elicited

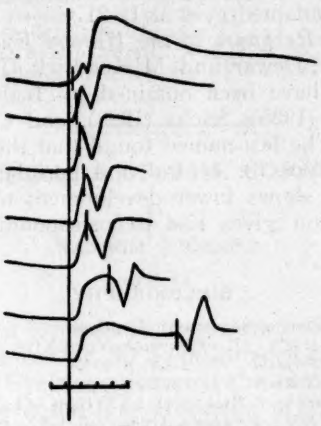

FIG. 11.

Effect of flashes (40σ) at various intervals after cessation of initial stimulus. Time marking $\frac{1}{10}$th second. (Granit and Riddell, 1934.)

by continuous stimulation of the same intensity (*e.g.*, 0·011 ml). As the frequency is increased the ripples become smaller and finally fuse. The fusion frequency varies directly as the logarithm of the brightness and the logarithm of the area over at least a hundredfold range (Ferry-Porter Law). The product of fusion frequency and latent period of ripples is approximately constant. The retinal process chiefly responsible for the ripples is PII, but there may be some interaction with PIII. At the fusion frequency the smooth curve of action potential approximates closely to that of a steady stimulus of half the intensity (Talbot-Plateau Law).

Granit and Riddell (1934) have analysed very thoroughly the effects of single flashes superimposed upon the eye at various intervals after the cessation of an initial stimulus of approximately constant duration. Fig. 11 shows the effect of a flash of about 40 σ superimposed upon the off-effect (*d* wave) of a light adapted retina.

In every case there is a negative dip (*a* wave), which is small at short and at long intervals and largest when at the top of the initial *d* wave. It would seem that intermittent ripples are produced by *a*, *b*, and *d* waves. At fast rates of stimulation fast ripples are produced by small *a* waves interrupting the off-effect produced by negative PIII returning towards the base line. At slow rates the waves become very brisk owing to increased negative dips followed by positive peaks (*b* waves) above the level of the off-effect.

In dark-adapted eyes fusion occurs at less than 6-8 flashes per second; in light-adapted eyes at 12-21.

The Electrical Response of the Human Eye. The first record was obtained by Dewar and M'Kendrick (Dewar, 1877); subsequent records have been obtained by Kahn and Löwenstein (1924), Hartline (1925), Sachs (1929), and Cooper, Creed, and Granit (1933). The last-named found that the curve of potential is of the same type for central and peripheral retina, but the peripheral curve shows lower development of potential. Intermittent stimulation gives rise to corresponding ripples on the curve.

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VARICELLA OF THE CORNEA

BY

RANSOM PICKARD

EXETER

THE occurrence of a corneal affection in varicella is a great rarity. Of the references given by Sir John Parsons in his work on the Pathology of the Eye none concerns the cornea, in the bibliography of an article by Dr. J. D. Rolleston on ocular complications in varicella (*Medical Chronicle*, p. 356, 1909) only two cases of varicella of the cornea are given, one of which is here cited (Oppenheimer's case). The writer of this note has not had access to the remaining case. Comby (*Traité des Maladies de l'Enfance, Grancher et Comby*, p. 380-1, 1904), writes:—

"J'ai vu aussi la varicelle atteindre . . . même la cornée, déterminant . . . kératite varicelleuse qui peut avoir sa gravité et laisser à sa suite une plaie indélébile." He does not state the number of cases he has seen nor does he give any details.

E. H. Oppenheimer (*Deutsche Med. Wochenschr.*, Vol. XXXI, p. 833, 1905), gives a detailed account of a case of varicella of the cornea. It occurred in a two-year-old girl. She and three siblings had had chicken-pox. The eye affection began in the left eye on the sixth day of the disease. In the outer lower quadrant, 3 mm. from the centre of the cornea, was a vesicle two pinheads in width, which had perforated. To the perforation was attached a small mucoid string. The upper lid and conjunctiva were much swollen and injected, and had muco-pus adherent to them. There was very little circum-corneal injection, although the bulbar conjunctiva was very red. The pupil was round, small, and reacted only slightly.

Very slight photophobia was present. In the course of the following day an ulcer formed, but by the eighth day of the eye affection (the fourteenth day of the varicella) this was covered with epithelium. Four days later a shallow, smooth hollow remained, a pin's head in size, without any sign of inflammation.

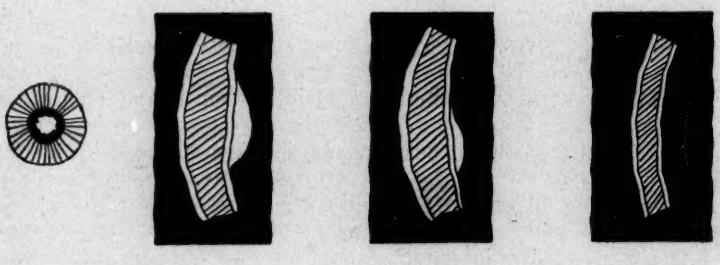
Oppenheimer adds a paragraph on the literature of corneal affections in varicella. He quotes Schmidt-Rimpler and Seitz as stating that occasionally corneal infiltrations occur, but points out that in a large number of books on children's diseases no mention is made of corneal affections in this disease.

Dr. A. Simey, of Exeter, for twenty-two years medical officer of Rugby School, tells me that, among thousands of cases of chicken-pox he saw there, no cases of keratitis occurred, although vesicles were occasionally seen on the conjunctiva.

This affection being so uncommon is the reason for recording the present case. The patient P. J., a boy, 10 years of age, was the last of a family of four children to develop varicella. The other three cases had been slight, the vesicles had the usual distribution, there being no herpetic grouping in any. This was also the case with P. J., who developed varicella on January 21, 1935. The right eye began to be irritable on February 11. He was seen by the writer on February 13.

The condition on February 18 was as follows:—A few varicella scabs still remained on various parts of the body and scalp. *Right Eye.* V. 6/24(2)n.i. Conjunctiva slightly pink. Iris normal but of sluggish reaction (at no subsequent period was there any synechia or deposit of pigment on the lens).

Cornea. There was a central opacity, well defined, grey in colour, with slightly crenated edge, circular in outline and in diameter equal to a quarter of the cornea. The slit-lamp showed the cornea to be swollen in the region of the opacity, not bulging forward but towards the anterior chamber. The section of the cornea was yellowish and mottled. On the posterior surface of



18/2/35 23/2/35 2/9/35

the cornea was a flat brownish-yellow mass, having a spongy free surface. The cornea elsewhere was normal. The left eye was normal.

Ung. Hyd. Flav. c. Atrop. was ordered to be used twice daily.

February 23. R. E. V. 6/18(1). Slit-lamp. Cornea still swollen, bluish-grey in the section with fine white granules. Near the upper edge the epithelium was raised ·04 mm. high over an area ·08 mm. in diameter (real, not apparent measurements). The mass on the posterior surface was smaller in all dimensions, above it had become a thin plate.

March 2. R. E. V. 6/12(1). The bleb on anterior surface and the deposit on posterior surface had gone.

March 9. Cornea of normal thickness and colour, except for a white anterior surface opacity and a brown sheen on the posterior specular reflex.

April 26. R. E. V. 6/5(3). Minute surface nebula, to the inner and lower side of corneal centre. With the exception of a small opacity in the middle layer at the lower part of the original site the substance of the cornea appeared normal.

September 2. R. E. V. 6/5(6). A minute surface nebula, about one-eighth of the cornea in diameter still exists. This cornea is now slightly thinner than the left unaffected cornea, but its curvature is normal.

It is to be noted that at no period was any thickening of corneal nerve fibres to be seen.

There are some resemblances between Oppenheimer's and the present case. In both the corneal affection began late; in his on the sixth day, in the present case twenty-one days after the onset of varicella. In both the affected area was small and well localized, the inflammation disappearing quickly and almost completely. Although no obvious iritis occurred in them at any time, the iris in each reacted slowly.

The main difference is that in Oppenheimer's case there was a vesicle which developed into an ulcer; in that now described there was only a microscopic bleb which did not perforate and soon disappeared.

The slit-lamp appearances showed that the keratitis involved the whole thickness of the cornea; and as usually occurs in the majority of cases of interstitial keratitis, the swelling was backwards towards the anterior chamber, not forwards. The brown exudate on the posterior surface of the cornea was confined to the site of the inflamed cornea, there was no other patch of keratitis punctata. It very closely resembled in colour and appearance a thin rubber sponge when the eye was first examined by the slit-lamp on February 18, seven days after the eye trouble had commenced. The final thinning of the cornea with no alteration in

its curve is noteworthy, the eye retaining full vision with no manifest error of refraction.

It is noteworthy that though the inflammation was severe with a good deal of exudate from the posterior surface of the cornea it yet remained localized, suggesting that it was caused by an organism having a limited growth whose course soon came to an end. It will be seen that in these respects the affection of the cornea is similar to that of the skin.

The question arises whether this might be considered as the sole manifestation of herpes of the first branch of the fifth nerve. A resemblance is the late period in which the corneal trouble arose, three weeks after the onset of the varicella; which is a very common time for herpetic affections of the cornea to begin. But there was no pain at any time, very little photophobia; while the rapid and practically complete recovery is very different from herpes. The simplest explanation is that it was a varicella eruption, occurring late because of the special structure of the cornea, a non-vascular and dense tissue which offers special difficulties to the transit of the virus.

A CASE OF CHLOROSIS WITH OCULAR COMPLICATIONS

BY

J. BRUCE HAMILTON

HOBART

IT is an undisputed fact that chlorosis as a disease died with the passing of the 19th century, and according to L. J. Witts[4] it has almost completely disappeared to-day. Its aetiology, and the reason for its disappearance are alike unknown, but Tidy[5] suggests that its disappearance may be attributed to the improved hygienic life of girls, to whom the disease was almost entirely confined.

The ocular complications of chlorosis have been occasionally recorded, and Foster-Moore[2] in 1925 was able to collect in the literature the records of 9 cases, reported between A.D. 1881, and the time of writing. But a request from me to the Librarian of the Royal Society of Medicine for photostatic copies of reports of all recent cases of chlorosis with ocular complications has resulted in only one record by Brazeau being found. In view of these facts I feel it incumbent on me to place on record a further case of chlorosis with ocular complications, and after reporting the case fully, I will make some comments on these complications.

Case Record

Mrs. J. B. 33 years of age. Home duties.

December 1, 1933. History. Born in England. Has been living in Australia 14 years. Two months ago while travelling in tramcar bumped head—no treatment. Six weeks ago bad pain in right side of head for 4-5 days, followed by double vision. Has never worn glasses. No history of general disability, except loss of weight.

Examination. Right vision, 6/4 and J.1. Left vision, 6/6 and J.2.

Muscle Balance. Distance, 30° esophoria, 1° left hyperphoria. Near, 15° esophoria, 2° left hyperphoria, no cyclophoria.

Right and left eyes. Slightly limited abduction. Pupils active to light and accommodation. No keratic precipitates. Three dioptres of swelling of both optic discs with complete star figure at each macula. No haemorrhages.

Refraction under mydriatic.

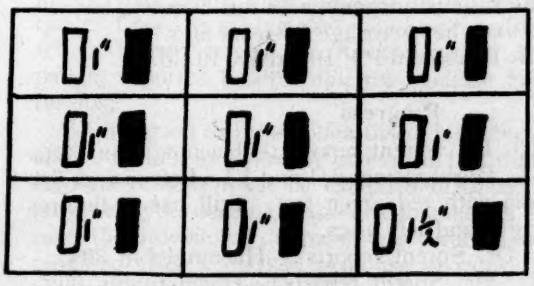
Right		Left	
	+ 1·5		+ 2·0
	+ 2·0		+ 2·5

Fields of vision. Right and left.

Form. Normal to 1 degree white, except for slight enlargement of both blind spots.

Colour. Normal to 1 degree red and green.

Diplopia Chart. At 1 metre.



= Red before Right.

= Green before Left.

Urine Test. S.g. 1006. Neutral. N.A.D.

Blood Count. Report from Dr. Walch:—

Haemoglobin, 45 per cent.; red cells, 3,510,000 per c.mm.; colour index, 0·64; white cells, 7,500 per c.mm.

	Per cent.	Per c.mm.	Normal per c.mm.
Polymorph. ...	72	5400	5025
Eosinophil. ...	9	675	225
Basophil ...	1	75	75
Lymphocytes ...	14	1050	1725
Monocytes ...	4	300	450
	100	7500	7500

Arneth class	1	2	3	4	5	Index
Normal ...	5	35	41	7	2	60
Patient ...	5	24	51	17	3	55

No nucleated red cells seen.

Red cells show slight anisocytosis, but are regular in shape and stain normally.

A marked anaemia of secondary type. The only distinctive feature in the differential count is eosinophilia.

(Signed) J. H. B. WALCH.

December 5, 1933.

Blood Test. Report from Dr. Walch.

Wassermann reaction, negative. Kline test, negative.

(Signed) J. H. B. WALCH.

December 5, 1933.

December 6, 1933. General examination. Report from Dr. Sprent.

"A stout woman giving a history of previous good health, with normally a sallow complexion. She now shows a characteristic greenish tint underlying her pallor. Her blood picture is not quite characteristic of chlorosis, but she has been taking a tonic containing iron. Chlorosis is uncommon in this country, but she has lived in London until her marriage 14 years ago.

I have given her Bi-Palatinoid Pil. Blaud gr. 10.t.d."

Progress

December 21, 1933. Dr. Sprent reports:—Haemoglobin 68%.

December 28, 1933. Right vision, 6/4 and J.1. Left vision, 6/6 and J.2. No diplopia with red green test. Still has 2 dioptres of swelling of the right and left discs.

January 4, 1934. Dr. Sprent reports:—Haemoglobin 80%.

January 25, 1934. Dr. Sprent reports:—Haemoglobin 90%.

March 7, 1934. Right vision, 6/4 and J.1. Left vision, 6/4 and J.1. Fields of vision. Right and left.

Form. Normal to 1 degree white. No enlargement of blind spots. Right fundus. Optic disc normal. Slight oedema of the macula.

Left Fundus. Optic disc slightly fuzzy. Remnant of ½ star between disc and macula.

April 6, 1934. Dr. Sprent reports:—Vomiting and headaches have returned. Is losing weight rapidly. Temperature normal. Haemoglobin 100%. Slight fuzziness of both optic discs with a remnant of nasal fan at each macula. Fields of vision. Right and left. Form. Normal to 1 degree white. Right vision, 6/4. Left vision, 6/4.

April 11, 1934. Admitted to hospital with provisional diagnosis of cerebral thrombosis in a blind area. Haemoglobin 100 per cent. X-ray of skull. Negative.

April 23, 1934. Lumbar puncture performed, but no details available, except W.R. of cerebro-spinal fluid was negative.

May 5, 1934. Discharged from hospital.

August 10, 1934. Report from Dr. Sprent. Patient well.

May 21, 1935. History. Lost 2½ stone in weight last year, but last 5 months has remained stationary. Is still taking tablets regularly. Occasional headaches. No further vomiting since lumbar puncture on April 23, 1934. Eye sight good.

Examination. Right vision, 6/4 and J.1. Left vision, 6/4 and J.1. Fundi. Right. Optic disc and macula normal. Left. Disc a little fuzzy nasally. Macula normal. Fields of vision. Right and left. Form. Normal to 1 degree white.

Comment

This is an undisputed case of chlorosis with complete recovery of eye signs following iron therapy, and the first feature that asserts itself in the eye signs is the lack of retinal haemorrhage. Knapp[3] and Foster-Moore,[2] both definitely assert that retinal haemorrhages are most unusual in chlorosis. In fact, Knapp quotes Leber as saying that it is questionable whether cases with retinal haemorrhages are true chlorosis. At no period did the patient reported here exhibit the smallest haemorrhage in either fundus.

With regard to the swollen discs, both these authorities quoted above, as well as Brazeau,[1] stress the fact that kidney disease and cerebral tumour must be excluded definitely and finally, before attributing the oedema of the disc to anaemia. In fact, in Brazeau's case, the provisional diagnosis was cerebral tumour, and was only modified after a marked response to iron. I think that the 18 months follow up in this case without return of swelling excludes both nephritis and cerebral neoplasm, and that the diagnosis of chlorosis is equally confirmed by the rapid decline of the swelling and diplopia, following the administration of iron.

The cause of the swelling is undoubtedly increased intracranial pressure, as proved by Pagenstecher, and quoted by Knapp.[3] The result of the lumbar puncture in this patient is unfortunately lacking, except for the Wassermann test, which was negative. But the patient asserts that she has had little or no headaches since the lumbar puncture; which I think is conclusive evidence that the cerebro-spinal fluid was under pressure.

With regard to the ocular palsy. This again is a rare occurrence in chlorosis, but cases with this complication have been recorded. This patient's diplopia rapidly disappeared with the exhibition or iron, and no trace of diplopia could be found 16 days after active treatment was commenced.

In conclusion I should like to acknowledge my indebtedness to Dr. James Walch for his report, and to Dr. James Sprent for his report and progress notes, over a period of 8 months. Also to Dr. Sprent for his comment on reading this proof, that chlorosis is not so dead in Tasmania as supposed, for in the past 25 years he has seen 6 cases of undoubted chlorosis in Hobart.

Summary

1. A case of chlorosis with ocular complications is reported.
2. Three dioptres of papilloedema was found in each eye.
3. All eye signs disappeared after intensive iron therapy.
4. The patient has been well and free from eye signs for 12 months.

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TREMULOUS LENS

BY

A. W. D'OMBRAIN, D.O.M.S(Lond.)

WEST MAITLAND, AUSTRALIA

A MAN aged 50 years was found to have an almost mature cataract in the right eye. The left eye was hypermetropic and with correction read 6/6. A month later the cataractous right eye developed irido-cyclitis. Atrophy, in patches, of the pigment layer of the iris was made out with the corneal microscope; also there were

present fluffy masses on the anterior surface of the collarette. Septic teeth were found and, after extraction of these, the irido-cyclitis cleared up completely under treatment.

Extraction of the now mature cataract was contemplated but on examination with the slit-lamp microscope, the lens was found to be tremulous. The iris was not tremulous so that there was no dislocation of the lens; the tremulousness of the lens, however, was of the same quality as that of a tremulous iris; on the slightest movement of the eyes, the lens trembled as if it were suspended on an elastic string.

Comment

I can only interpret this phenomenon as being due to fluidity of the vitreous; the attack of irido-cyclitis was a severe one and at its conclusion left the eye with a slightly softer intra-ocular tension than the other eye. I have not found any reference to tremulousness of the lens in such literature as I have been able to procure.

TUBERCULOSIS OF THE CHOROID ASSOCIATED WITH GENERALIZED MILIARY TUBERCULOSIS*

BY

FREDERICK TOOKE

MONTREAL, CANADA

FOR many years past the allegiance of oculists has been challenged. The question at issue has been, which has provided the greater source of scientific satisfaction, a foundation upon which a future superstructure of a complete ophthalmic expression can ultimately be raised, the Eye Hospital, Infirmary, or Institute, or the Ophthalmic Clinic as an integral unit in the various elements which form the regularly standardized or constituted hospital of to-day.

For the practising ophthalmologist, and much more for the self-styled ophthalmic surgeon, a great deal is to be said for the Infirmary in supplying in quantities operative material without which no surgeon can approach his subject with assurance, develop the delicacy and refinement of a surgical technique which the specialty imperatively demands, or in an entirely ethical sense by introducing him to the community at large through the medium

* Read before the American Ophthalmological Society, July, 1935.

of his public service. The same statement holds true regarding the diagnosis and treatment of what we have been pleased to term external diseases; often the expression of some remote systemic disorder, unfortunately only too frequently regarded by the ophthalmologist in a surgical sense and treated as such.

But our ophthalmologist must to-day be much more than an ophthalmic surgeon. He must approach the solution of his sometimes extremely complex riddle by more devious channels of investigation and interrogation than is the case possibly in any other department of modern medicine. For such an individual who as a student demands a completely rounded out enquiry into a relatively small number of cases, his sources of information must be open and near at hand.

With such a preamble as an oculist attached to a department in a general hospital, may I presume to argue that a great deal of most valuable and interesting scientific information is very frequently provided for the seeker after truth, from the various elements of a hospital in which a Department of Ophthalmology exists. The consideration of the case which I am about to report would bear out such an argument. The almost daily observation of the patient by my associates in the service supporting my observations would strongly emphasize the stand I have presumed to take.

J. D., a male infant of thirteen months was admitted to the Paediatric Department of the Royal Victoria Hospital, Montreal, December 22, 1934, and died January 14, 1935, twenty-three days later. Three days prior to admission the child had appeared to be in his usual good health, but he suddenly became irritable and restless. He refused part of his breast feeding and his sleep was broken. There had been three attacks of vomiting, and the patient seemed to be feverish. During this period he had suffered from a series of convulsions which were invariably induced when he was disturbed or moved; one of these occurred at home lasting for three minutes when his physician was in attendance. The child became quite pale, the eyes and head were turned to the right, the body was limp, and the fingers were extended. During the convulsion and after it Chovestek's sign was present; between the convulsions the patient was relatively happy, comfortable and contented. He laughed, played, and did not appear to be sick.

From a personal and family history there did not appear to be any predisposition towards tuberculosis from the child's forebears, but an old friend, who was very suspiciously tuberculous had frequently been in attendance on the child when the mother required assistance.

On admission to the hospital another characteristic convulsion

took place almost at once, after which the temperature registered 101°. The pupils were regular, and of equal size, and reacted to light; the fundi were normal. The anterior cervical glands were bilaterally enlarged, while the posterior cervicals were small and shotty. An examination of the chest showed it to be symmetrical with regular movements on respiration; the percussion note was resonant throughout, breath sounds were clear and no

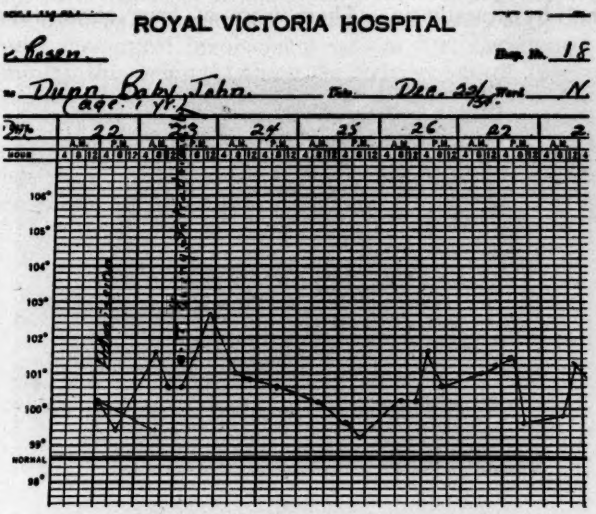

FIG. 1.

Typical tuberculin reaction following intradermal injection of 1/10 mg. O.T. Temperature after its initial fall gradually rose to 105° at exitus.

râles were heard. The spleen, however, was enlarged and soft. The tendon reflexes were generally increased, while the abdominal reflexes were absent. The tonsils were enlarged.

A lumbar puncture was performed and an absolutely clear fluid was withdrawn containing only two cells per c.mm. The Pandy test was negative. There was no stiffness of the neck and Kernig's sign was absent. The day following admission 1/10 mgm. of O.T. was administered intra-dermally with a typical local and general reaction (Fig. 1), while an X-ray photograph of the chest showed the lungs to be peppered or mottled with innumerable miliary tubercles (Fig. 2). Four days following admission a lumbar puncture was again performed when eight c.c. of clear fluid were withdrawn; there were only six cells per c.mm., and pressure

could not be recorded. Five days following admission sputum was recovered from stomach washings and tubercle bacilli were demonstrated.

When I was called upon to examine the fundi at this date I detected a crop of characteristic tubercles of the choroid in each eye. A maintained examination could not possibly be carried out owing to the child's age, on account of its precarious health, and also because the introduction of bright light created a retinal hyperaesthesia. There were at least seven tubercles

FIG. 2.

X-ray photograph of chest showing generalized miliary tuberculosis of lungs.

in the right fundus and five in the left; probably more. They were situated at the temporal sides of the disc margins just before one arrives at the macular area; there was one tubercle observed further to the temporal side and above and beyond the macular area in the right fundus oculi. The tubercles measured from one to two millimetres in diameter and could best be described as occurring as a cluster or a group. They did not select the tissue underlying the bifurcation of the retinal vessels as is so frequently the case; indeed the retinal vessels were absolutely uninvolved in the clinical picture. The disc surfaces were of good colour and their margins were sharply outlined, quite unlike the blurred margins of discs frequently associated with choroidal tubercles occurring after an advanced tuberculous meningitis has been established clinically.

The two lumbar punctures carried out up to date would appear to have excluded the meningeal factor. On January 8, or six days prior to death, a lumbar puncture was again performed when three

to four c.c. of clear fluid were withdrawn showing only ten cells per c.mm. Pandy negative. Four days prior to death there was no stiffness of the neck. The patient's eyes were repeatedly examined up to two days prior to exitus and the picture originally revealed in the fundi showed little if any variation from that which I have already described. Temperature at the time of death rose to 105°.

A general autopsy was performed and revealed a diffuse tuberculous involvement of all the parenchymatous organs. There was a caseous focus demonstrated in a right peribronchial lymph gland. Grossly meningeal involvement was at first questioned, but on examining the meninges along the Sylvian fissure, tubercles were noted; they would appear, however, to have been but few. What was of greatest significance was that on sectioning the brain there was revealed an area of caseation involving the cortex of the left parietal region; a primary focus ante-dating the appearance of the tubercles on the meningeal capsule or sheath. Autopsy also

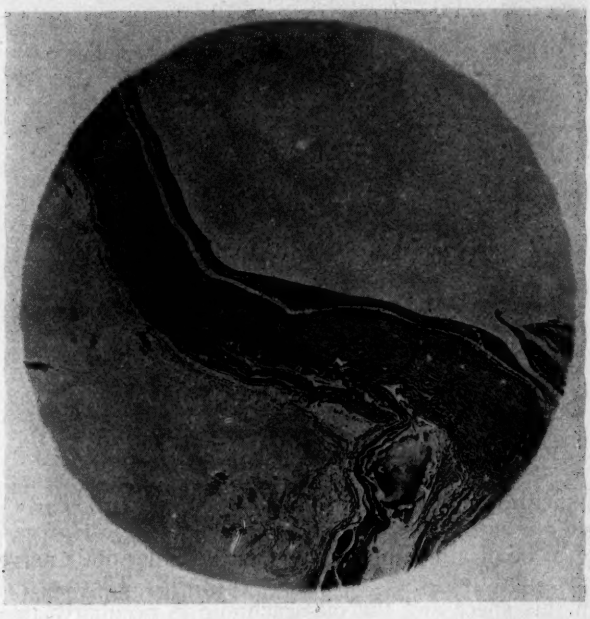

FIG. 3.

Low power magnification. Shows at least three tubercles or tuberculous foci. Absolute absence of involvement of vaginal space and optic nerve sheath.

revealed extensive miliary tuberculosis involving the lungs, liver, spleen, kidneys, adrenals, thoracic and abdominal lymph glands, pancreas and myocardium, with exudative ulcertive tuberculous enteritis.

The posterior half of each eye was excised and sections were made in paraffin.

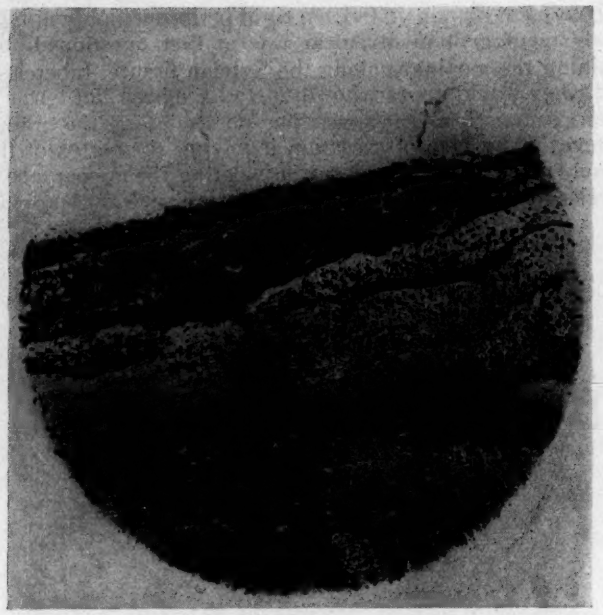

FIG. 4.

High power magnification. Longitudinal section of optic nerve showing infiltration of nerve fibres through contact with involved pia-arachnoid.

The picture which attracts one's attention particularly is the absolute absence of association of any pathological process purporting to be of a meningeal character in either optic nerve itself or in the subdural or vaginal space (Fig. 3). There are no free cells in this space and the pia-arachnoid is peculiarly free from an associated leucocytic infiltration. I am showing by way of contrast a section of a blurred optic nerve occurring with a choroidal tubercle which appeared in a case of advanced tuberculous meningitis shortly before death (Fig. 4). Here, microscopically,

one can note the unquestionable infiltration of the outermost nerve fibres obviously associated with the pathological changes in the meninges or in the cerebro-spinal fluid contained within the vaginal space.

In the sections obtained and considered seriatim one can see what can better be described as four tuberculous areas rather than

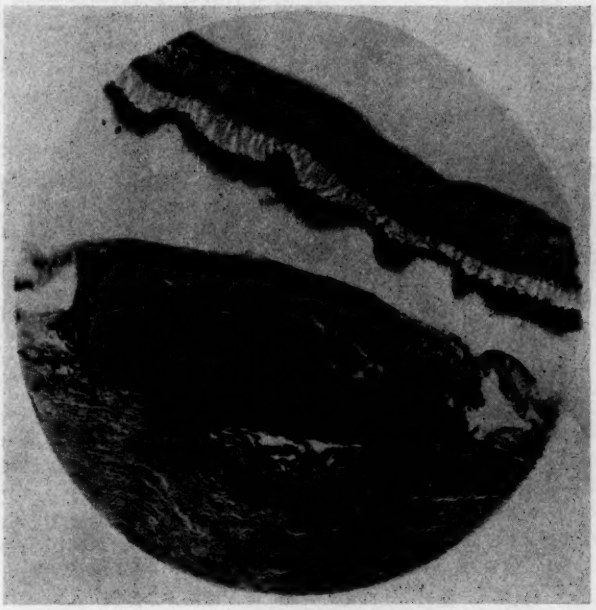

FIG. 5.

High power magnification. Recent tubercle with dense circumscribed infiltration of leucocytes encapsulating area of tuberculous tissue.

what one is accustomed to acknowledge or recognize as actual tubercle formations (Fig. 3). In a true tubercle with Bielschowsky's stain one would expect to find a trabecular formation of the tubercle proper formed by collagen fibres, the whole zone to be surrounded by a limiting capsule of lymphocytes and fibrinous exudate including or walling off the other well recognized component parts of the tubercle proper. Here such a picture is not afforded one, and there is no such thing as a sharply limited zone of peri-tubercular infiltration discernible (Fig. 5). In fact the

acute phase of an intense perivasculitis with the establishment of perivascular tubercles about the capillaries is but very feebly in evidence. The choroidal vessels, it is true, are decidedly dilated but one cannot note any degree of perivasculitis. There are quantities of epithelioid cells, giant cells without number, and the condition of granulation tissue with coagulation necrosis which

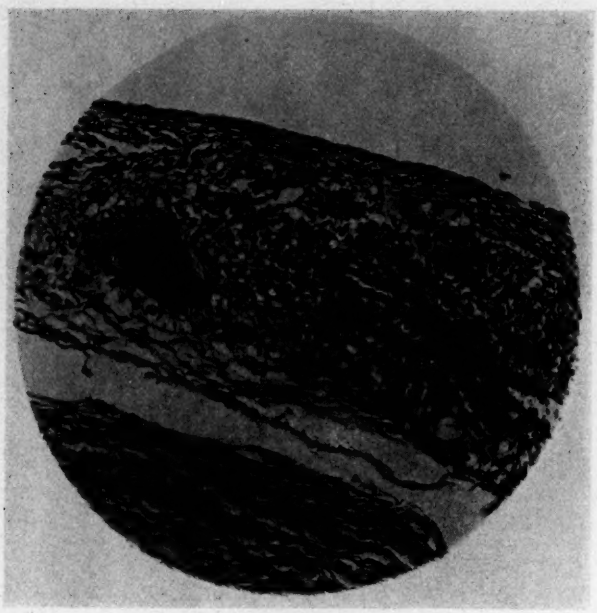

FIG. 6.

High power magnification. Shows chronic tuberculous formation without encapsulation as in recent tubercle formation. Epithelioid cells, coagulation necrosis and giant cells well marked. Very little perivasculitis.

would indicate an established rather than an incipient lesion still in the process of extension, but relatively a chronic rather than an acute picture pathologically regarded. The whole process was limited within the membrane of Bruch (Fig. 6). Tubercle bacilli were demonstrated in the tissues.

In reviewing the clinical and pathological facts in so far as I have been able to establish and record them, the question arises how far are we in error in associating the establishment of

tubercles of the choroid with a co-existing problematic, or for that matter even with an actually existing, tuberculous meningitis rather than with the underlying early or miliary type of a generalized tuberculous infection carried by the blood stream to various foci prior to an involvement of the meninges. The choroidal tubercle may be no more a complication or an extension of a meningeal tuberculosis than it is one of a tuberculosis involving the peritoneum, pleura, pericardium or any other membrane enclosing a serous cavity within which an active tuberculous process may be, clinically at least, evident and manifesting expression.

Rich and McCordock (J. Hopkins Bulletin, 1933) in their excellent article have reported upon the gross pathological findings of eighty-two cases of tuberculous meningitis not necessarily associated in so far as I have been able to learn with tubercles in the choroid such as those which I am reporting. A review of their conclusions has a very direct bearing on the case under consideration. In the vast majority of their cases, in seventy-seven out of eighty-two, older tuberculous foci were detected in the adjacent brain tissue, the bones of the cranial cavity, or about the spinal cord, or in the meninges themselves. In other words, the condition of meningitis was not brought about through the blood stream but rather through direct contact of an old caseous or necrotic area with the meninges or with the cerebro-spinal fluid adjacent to it. The same argument holds true regarding the cerebro-spinal fluid affecting the fibres of the optic nerve in the section that I have already shown (Fig. 4). It is but natural to enquire regarding the origin of the local areas of necrosis assumed to be responsible for the meningitis as in the present case the tuberculoma of the left cerebrum; undoubtedly it was the result of the early dissemination of bacilli which so frequently follows upon a primary infection. No matter what the site of a progressive lesion may have been, let us assume the peribronchial glands, a scattering of bacilli to distant organs through the blood stream has been generally demonstrated; such a dissemination resulting oft-times in sparsely scattered tuberculous masses, "solitary tubercles" of varying size with or without competent incapsulation which I demonstrated to have occurred in the choroid (Fig. 6). Many of these foci have become sterile; in others bacilli remain alive as I have demonstrated in my section; but in the presence of acquired resistance or relative chronicity their power of multiplication has been held in abeyance and the lesion has remained quiescent as was most certainly the case during the last ten days of this patient's life.

Early dissemination, which occurs before an acquired resistance has been established following a primary infection, results in areas

of caseation which extend to infect the meninges in infancy and early childhood.

Miliary tuberculosis has so commonly been associated with tuberculous meningitis in the very young that the latter has long been assumed to be simply a manifestation of the former. One should stress not so much the coincidence of a meningitis with miliary tuberculosis as the coincidence of a meningitis with a low degree of acquired resistance. In brief, again to quote Rich and McCordock, the sequence of events is not (1) primary lesion, (2) meningitis occurring as a part of a miliary tuberculosis; but rather (1) primary lesion, (2) dissemination of bacilli leading to formation of caseous foci in structures adjacent to the meninges with or without frank miliary tuberculosis, (3) discharge of bacilli from these foci into the meninges causing meningitis. These authors have shown that isolated tubercles of the brain or meninges arising as part of a slowly progressive miliary tuberculosis may at times, as in this particular instance, resolve into caseous foci large enough to infect the cerebro-spinal fluid by discharging large numbers of bacilli directly into it. One must regard the development of a meningitis as a fortuitous event, depending not only upon the existence of a generalized miliary tuberculosis but upon the chance extension of infection from an established caseous focus which happened to develop adjacent to the meninges.

Summary

1. Tubercles of the choroid are established as a haematogenous infection in a condition of a generalized miliary tuberculosis.

2. Tuberculous meningitis results from contact with necrotic foci from adjacent tissues earlier involved through the blood stream.

3. Tuberculous meningitis is a late rather than an early manifestation of the course of the disease.

4. Tubercles of the choroid in certain cases are coincident rather than complicating manifestations of tuberculous meningitis.

5. Tubercles of the choroid may exist not because of, but in spite of, tuberculous meningitis.

GLUTATHIONE IN THE BLOOD IN SENILE CATARACT AND OTHER OCULAR CONDITIONS

BY

DOROTHY ADAMS CAMPBELL

BIRMINGHAM

Introduction

IT is well known that the human lens is rich in glutathione and that this substance disappears totally from the lens in senile cataract. Glutathione is remarkable in being the most labile sulphur-containing substance in the body. It is, as we know, closely associated with tissue respiration, which assumes special importance in a non-vascular tissue such as the lens. The relation of glutathione to the general sulphur metabolism of the body has never been determined (Lewis), either in the normal subject or in disease. As a first step towards this investigation it was thought worth while to estimate the blood glutathione in cases of senile cataract and other ocular disease, the range for blood glutathione in the normal being already well known. In addition, in some concurrent investigations (Bourne and Campbell), a study was made of the sulphur metabolism in the same types of patients, by giving diets of known sulphur value and estimating the corresponding sulphur excretion in the urine. The figures obtained from the two series of experiments provide an interesting first record of the relation of blood glutathione to sulphur metabolism, particularly in senile cataract as compared with other ocular disease. In actual fact, the sulphur metabolism in these cases was not found to be abnormal, as might have been expected.

Historical

The bulk of the glutathione present in the blood is in the reduced form, which varies from 25-41 mgm. per 100 c.c. (average 34), while the oxidised form varies from 3-11 mgm. (Woodward and Fry). This is in agreement with the earlier observations for reduced glutathione in the normal subject—Benedict and Gottschall 30-48, Mason 36-58, Achard, Levy and Guthmann 36-58 and Groen and Meyer 40 mgm. It is wholly contained in the corpuscles, and if considered in relation to the blood count, the factor, reduced glutathione, red cell count, ranges from 4·9-9·1 (Woodward and Fry).

Little is known as to the variation of glutathione in disease. Achard, Levy and Guthmann (1932) found a slight decrease in anaemia, diabetes, neoplasm and tuberculosis, while Giordano and

Battistini (1933) found no variation in a number of diseases. Senturia (1934) states that there is no variation from the normal in cases of arthritis, while Loomis and Bogen (1934) found a slight increase in the reduced form, and a decrease in the oxidised, in tubercle, which they ascribed to defective aeration of the blood. Oberst and Woods (1935) recently estimated the blood glutathione in 66 women.

The only figures available for patients with ocular disease, are those of Vele (1933), who employed an older and rather less accurate method of estimation. For normal patients, she found a range of only 13·5-24·5 mgm. per 100 c.c. (average 18·25), whereas in a large variety of ocular disease she found an increase in every type of case, ranging from 15-60 mgm. She states that there was an accompanying rise in the red cell count, and whereas the factor GSH/r.b.c. was 4 in the normal, it was 6-8 in ocular disease, and even higher in keratitis.

Experimental

The usual methods for the estimation of glutathione were based on the reduction of iodine, but in the last few years, reducing substances other than glutathione have been shown to be present in the tissues, *e.g.*, thionine and ascorbic acid (Vitamin C), which interfere with the estimation. Thus in the ox lens, 1/5 of the total reduction of iodine by a trichloracetic acid filtrate, formerly ascribed wholly to glutathione, is due to Vitamin C, and in the case of a human lens from a 30-year-old subject, 28 per cent. of the reduction was due to Vitamin C. (Euler and Martius.)

For the present series of estimations, therefore, the method of Woodward and Fry (1932) was used, in which the interference from other substances is very small. Thus the amount of thionine in the blood could cause at the most an error of 3 mgm. per cent., while Vitamin C is present in the reduced form to the extent of only 0·5 mgm. per 100 c.c. blood. One slight variation was made in the method, *viz.*, in order to obtain the figure for total glutathione, reduction of the solution with zinc was carried out at 37°C. instead of at room temperature.

In every case, samples of venous blood were analysed immediately after withdrawal, the blood being taken not less than two hours and a half after a meal. As the range of glutathione has been adequately demonstrated for the normal, the estimations on patients with senile cataract were compared with those from cases of other ocular disease, exclusive of any condition affecting the lens. In cataract patients, the blood was taken at a time when there was no active absorption of lens matter in progress, *i.e.*, after a preliminary iridectomy when the lens had not been

disturbed, or some time after an extraction when no residual cortex was present.

With very few exceptions the patients were under observation in hospital, and it was possible to study a number on a strictly balanced diet of known calorie value, for three successive days. A comparison of the blood glutathione was made in the two classes of patients (*a*) senile cataract, (*b*) ocular disease exclusive of cataract, as follows:—

(1) On ordinary hospital diet, uncontrolled.

(2) On controlled normal diet of known calorie value and sulphur intake.

(3) In relation to the red cell count, on ordinary diet.

(4) In relation to the red cell count, on controlled high protein, low protein and normal diets of the same calorie values.

An example is given of the controlled normal diet of 2,347 calories which was most used, and details of the high and low protein (*i.e.*, high and low sulphur) diets can be found elsewhere, in a full description of the investigation of the sulphur metabolism in these patients (Bourne and Campbell). The results are summarised in the following tables. The abbreviations "GSH" and "GSSG" are used to signify reduced and oxidised glutathione respectively. The sulphur and protein values of foodstuffs were taken from Sherman's "Chemistry of Food and Nutrition," 1933.

TABLE I

BLOOD GLUTATHIONE, REDUCED AND OXIDISED, IN PATIENTS ON ORDINARY HOSPITAL DIET

Patient	Sex	Age	GSH (mgm. per 100 c.c. blood)	GSSG (mgm. per 100 c.c. blood)	Clinical details
C. C. ...	M.	56	50·15	2·40	Senile cataract
W. S. ...	M.	71	38·95	3·58	
M. T. ...	F.	71	35·12	2·29	
L. S. ...	F.	75	53·37	1·83	
H. A. ...	M.	75	51·21	2·16	
			45·76	2·45	Average
C. B. ...	F.	9	39·87	0·00	Strabismus
G. C. ...	M.	9	32·82	3·06	Strabismus
P. ...	F.	33	36·19	4·91	Perforating injury, L.E.
H. B. ...	M.	43	41·10	0·91	Specific iritis, R. and L.
L. F. ...	F.	52	38·95	6·14	Lacrimal abscess
A. C. ...	M.	58	45·70	1·23	L. excision, choroidal sarcoma
J. L. ...	M.	62	46·01	0·91	Glaucoma, R. and L.
E. J. ...	F.	71	36·80	3·99	Secondary glaucoma, L.E.
			39·68	2·84	Average

Range—35·12–53·37 1·83–3·58 Senile cataract
32·82–46·01 0·00–6·14 Non-cataract cases.

TABLE II

BLOOD GLUTATHIONE IN PATIENTS ON CONTROLLED NORMAL DIET

Patient	Sex	Age	Diet Calories	GSH mgm. per 100 c.c. blood	GSSG	Clinical details
H. S. ...	F.	43	2495	39·79	6·22	Senile cataract
A. McV.	F.	51	2347	36·80	—	
T. H. ...	M.	54	2347	36·96	0·46	
G. W. ...	M.	55	2347	38·34	1·53	
C. B. ...	F.	57	2347	37·42	1·84	
M. C. ...	F.	57	2178	46·01	6·41	
J. B. ...	M.	65	2347	41·01	3·05	
H. S. ...	M.	67	2347	41·71	4·30	
G. S. ...	M.	68	2347	49·69	3·99	
J. W. ...	M.	69	2347	46·01	0·89	
G. L. ...	F.	79	2393	44·01	—	
				41·61	3·19	Average
M. P. ...	F.	29	2495	45·39	—	Detachment L. retina
E. O'D.	F.	31	2347	41·71	12·89	Keratitis R.E.
L. H. ...	M.	33	2347	42·94	9·59	Strabismus
M. P. ...	F.	47	2347	40·82	2·12	L. excision for injury
N. ...	M.	50	2347	42·01	2·78	Detachment R. retina
J. J. ...	F.	59	2347	45·39	2·46	R. corneal ulcer
M. H. ...	F.	59	2147	45·09	1·23	Iritis L.E.
				43·34	5·18	Average

Range—36·80–49·69 0·46–6·41 Cataract
40·82–45·39 1·23–12·89 Non-cataract

N.B.—The same diet was continued in each patient for three successive days for the metabolism experiments, the blood sample for glutathione being taken on the second day.

The diet of 2347 calories contained 66·94 gm. protein, 1·935 gm. sulphur, the other diets differed from this only very slightly.

TABLE III

BLOOD GLUTATHIONE IN RELATION TO THE RED CELL COUNT IN PATIENTS ON ORDINARY HOSPITAL DIET.

Patient	Sex	Age	Red cell count	GSH	$\frac{\text{GSH}}{\text{r.b.c.}}$	Clinical details
H. ...	M.	—	4,450,000	32·80	7·37	Senile cataract
W. H. ...	M.	66	5,500,000	36·80	6·60	
S. K. ...	M.	67	4,600,000	42·33	9·20	
J. L. ...	F.	68	5,050,000	47·23	9·35	
W. R. ...	M.	70	5,550,000	53·98	9·72	
A. C. ...	F.	74	5,200,000	47·85	9·19	
E. B. ...	F.	77	5,150,000	39·57	7·66	
				42·98	8·44	Average
M. F. ...	M.	10	4,100,000	33·74	8·02	Interstitial keratitis
M. V. ...	M.	10	3,750,000	37·11	9·89	,, ,, W.R. + ve
W. ...	M.	20	4,750,000	36·19	7·61	,, ,, W.R. + ve
F. F. ...	M.	32	5,600,000	54·29	9·69	Scleral wound, R.E.
E. W. ...	F.	40	4,700,000	35·12	7·40	Detachment of retina
H. M. ...	F.	43	5,600,000	46·93	8·38	,, ,,
M. W. W.	F.	62	4,300,000	46·93	10·93	Secondary glaucoma, L.E.
A. M. ...	M.	69	5,600,000	58·29	6·68	Chronic glaucoma, R. and L.
				42·07	8·56	Average

Range—32·80–53·98 6·60–9·72 Cataract
33·74–54·29 6·66–10·93 Non-cataract

TABLE IV

BLOOD GLUTATHIONE IN PATIENTS ON GRADED AND CONTROLLED DIETS

Patient	Sex	Age	Diet	Red cell count	GSH	GSSG	GSH / r.b.c.	Clinical details
H. T....	M.	22	H.P.	5,600,000	35·12	5·98	6·27	Perforating injury R.E.
			L.P.	—	35·27	2·46	6·42	
			N.	5,350,000	34·04	—	6·36	
G. G....	M.	50	L.P.	4,450,000	37·11	0·31	8·33	Glaucoma, R. and L.
			N.	—	36·50	—	8·20	
G. S....	M.	68	L.P.	5,650,000	42·33	2·66	7·49	Senile cataract, R. and L.
			N.	—	38·95	3·07	6·89	
G.W....	M.	72	H.P.	5,100,000	29·44	8·90	5·77	Senile cataract, R. and L.
			L.P.	—	37·41	3·69	7·07	
			N.	5,200,000	38·34	4·60	7·03	
D. T....	M.	73	H.P.	3,250,000	26·68	4·00	8·21	Senile cataract, R. and L.
			L.P.	—	26·07	1·61	8·02	

N.B.—Each diet was continued for three successive days, one day's interval being allowed between the change from one diet to the next. Blood was taken on the second day for estimation and cell count.

Daily diet:	*Protein*	*Sulphur*	*Calories*
High protein (H.P.) ...	107·42 gm.	3·987 gm.	2325
Low protein (L.P.) ...	33·23	0·931	2328
Normal (N.)	67·87	1·863	2261

TABLE V

EXAMPLE OF CONTROLLED NORMAL DIET OF 2347 CALORIES PER DIEM

Food	Ounces	Gm. Protein	Gm. Sulphur
Bread	8	20·61	0·22
Butter	2½	0·63	0·006
Egg	2	7·50	0·109
Milk	10	9·24	0·094
Sugar	2	—	—
Beef	4	21·17	1·283
Potato	6	3·36	0·050
Cabbage	6	2·68	0·099
Apples	8	0·89	0·013
Cream	1	0·70	0·008
Marmalade	1	0·16	—
	Total	66·94	1·935

distributed as follows:—

Breakfast—Bread 2 oz., butter ½ oz., 1 egg boiled.

Lunch—Bread 1 oz., butter ¼ oz.

Dinner—Beef 4 oz. (sirloin steak, minced raw, weighed and cooked with water), potato 6 oz. (weighed raw after peeling, steamed), cabbage 6 oz. (weighed raw, and steamed), apples 8 oz. (weighed after peeling, stewed with 1 oz. sugar), cream 1 oz.

Tea—Bread 3 oz., butter ¾ oz., marmalade 1 oz.

Supper—Bread 2 oz., butter ½ oz., milk 5 oz.

Fluid not less than 2½ pints as tea or coffee allowed for the day, together with sugar 1 oz., and milk 5 oz.

Summary of Results

(1) There is no marked variation in the reduced glutathione of the blood, with age or diet, or in senile cataract as compared with other ocular conditions. The range for all cases of ocular disease is within the normal.

(2) In comparison with reduced glutathione, the oxidised glutathione shows considerable variation, especially in non-cataract cases, but no significant change.

(3) The factor GSH/r.b.c. is not raised above the normal value in ocular disease, and shows the same range in senile cataract as in other ocular conditions, including active keratitis (*cf.* Vele).

Comment

Although these results are negative, it is particularly interesting to find that the blood glutathione, like the blood calcium, remains constant over a certain range in a variety of conditions of age and disease. Moreover, in ocular disease, it appears to be unaffected by the sulphur metabolism of the body, even when there is a large variation in the amount of sulphur which is being ingested. This is probably true also for normal subjects.

In conclusion, I am indebted to the Birmingham and Midland Eye Hospital for a research grant covering the cost of these investigations. I am grateful to my colleagues on the staff for allowing me free access to their cases, and to Miss Marshall for doing the blood-counts.

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THE LOCALIZATION OF THE RETINAL HOLE

BY

J. A. VAN HEUVEN

UTRECHT

THE accurate localization of a point seen ophthalmoscopically in the fundus and its projection on the outer surface of the sclera is difficult but of vital importance in the surgical treatment of retinal detachments.

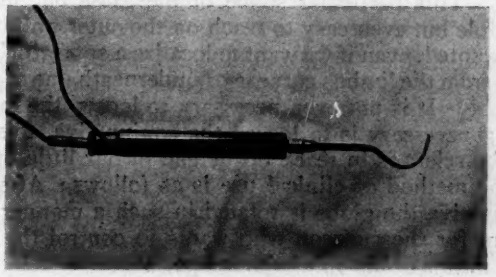
Several methods have been elaborated to attain accuracy and the fact that different surgeons still use various methods shows that none of these supplies every want.

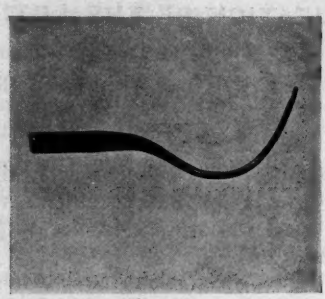
The different methods used are summed up in the third edition of Duke-Elder's Recent Advances as far as 1934. During the year 1935 some papers have been published about this problem, among which I want especially to mention that of Dr. Strampelli (Paris Congress, 1935).

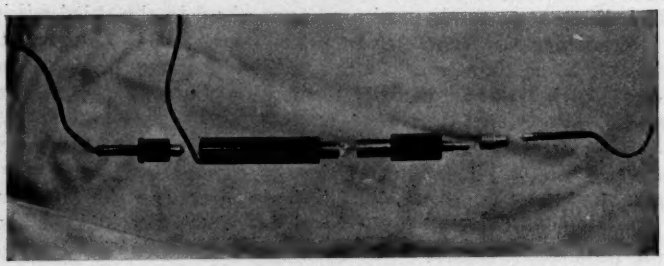
As evidently the question is still unsettled, I want to describe a method which I have been using for some time.

In principle a small pencil of light is placed on the outer wall of the sclera and moved until the right spot is reached. This is made possible in a better way than before by means of a special shape of transilluminator.

A glass bar of a thickness of 3 mm. is pulled out and bent in three different dimensions (see photo). At one end there remains a thickening in the form of a flattened knob, the stem varies in thickness between 2 to 1·5 mm. This little bar is now first silvered and afterwards varnished with a lacquer which stands boiling. Furthermore, it has a very narrow lumen, through which a wire of platinum is drawn, which just projects out of the flattened knob. Over a small surface with a diameter of about 1 mm. round about the spot where the wire projects out of the glass bar the cover of silver and varnish is scratched off.







By means of a screw-tap the other end of the bar is made to fit exactly an electric ophthalmoscope after Simon, which is very commonly used on the continent. The wire of platinum which runs through the bar is led out of this separately and well insulated.

The light of the ophthalmoscope is now totally reflected by the silvered walls of the bar and finally is stored in the flattened knob out of which it is only allowed to come through the small opening where the cover is scratched off. Here it appears as a small but rather intensive pencil of light.

The fitting on the ophthalmoscope does not allow any light to come out. This particular shape of the glass bar makes it not only possible but even easy to reach on the outer wall of the globe any spot wanted, even if we want to localize a spot which is situated rather far from the limbus corneae or underneath one of the muscles of the globe. It is not even necessary to loosen the muscle. The only thing necessary is to loosen the conjunctiva, this done, any spot on the sclera is in reach of the end of our little bar.

Now the method of clinical use is as follows: After loosening the conjunctiva the globe is rotated in such a manner, as is most convenient for the surgeon who wants to control the hole in the retina by means of his ophthalmoscope.

Now the transilluminator is pressed against the wall of the globe somewhere about the right spot, which may be done by any assistant or nurse. Whilst ophthalmoscoping, the surgeon gets in view the light of the transilluminator which penetrates the sclera. He is then able to direct this light in such a way that it coincides with the spot of the retina which must be treated. If, finally, this is the case the surgeon allows a diathermic current to run through the wire of platinum by means of a foot-circuit-closer. By doing so a small burned spot on the sclera will mark the exact spot of the centre of his pencil of light. The apparatus is now taken away and the therapeutic diathermic current is applied, either surface coagulation alone or combined with perforation, depending on the case. After treatment the instrument is useful for control of the results.

Why not go one step further and make a sort of "Universal instrument," by which it would be possible to apply at once the necessary amount of diathermic current at the same time as marking the exact spot?

This question was asked by Professor Weve when I showed him the apparatus. Weve made such an instrument in which the wire of platinum was made movable so that it could also be used for perforation, but for this purpose it was necessary to change the particular shape of the glass bar and to make it much thicker and less curved. By doing so it becomes less practical. After some experiments we abandoned this idea. The apparatus should only be used for marking, not at the same time for therapeutic purposes.

I have been using this instrument in private work for over a year and have obtained satisfactory results.

ABSTRACTS

I.—GENERAL MEDICINE

(1) **Kreibig, W. (Vienna).—Conjunctival lesions in leukaemia. (Ueber Bindehautveränderungen bei leukaemischen Erkrankungen).** *Zeitschr. f. Augenheilk.*, Vol. LXXXIV, p. 120, 1934.

(1) **Kreibig** holds that apart from orbital and retinal infiltration, leukaemia also gives characteristic conjunctival lesions. They are of particular significance as they may be the first manifestation of the disease. His article is based on isolated cases reported

in the literature and on five cases of his own. He distinguishes three clinical types: (1) Most commonly the infiltrates affect the fornices; diffuse, oedematous thickening is present in which follicles may be distinguished; only exceptionally does this thickening spread on to the bulbar or tarsal conjunctiva.

(2) In some cases the bulbar conjunctiva is mainly affected by this diffuse thickening, with the fornices relatively free; the swelling is most marked at the limbus, and in these cases exophthalmos is generally present. The infiltrate spreads backwards in and around the capsule of Tenon.

(3) A nodular form in which brown or bluish-red clearly demarcated nodules are seen in the conjunctiva, especially in the fornices. These particular changes occurred only in a leukaemic lymphadenosis.

Attention is drawn to the orbital lymphatics as a channel for spread and to the fact that conjunctival changes are not seen in the myelogenous form of leukaemia.

ARNOLD SORSBY.

(2) **Cogan, D. (Boston).—Uveoparotid fever.** *Amer. Jl. of Ophthal.*, Vol. XVIII, 1935.

(2) **Cogan** reports a case of uveo-parotitis in a woman, 29 years of age, who had suffered from mumps 6 years previously and in childhood had tuberculous cervical glands with sinuses, now healed. Facial paralysis of toxic origin, dental abscesses, and Vincent's infection of the mouth were other clinical signs of interest besides those typical of the disease.

The literature of this subject is briefly reviewed and the aetiology, clinical features and sequelae discussed. The author is in favour of the theory that tuberculous disease of a fibrosing, non-caseating, lymphadenopathic type is an aetiological factor although he admits that tubercle bacilli have never been isolated from the affected lymph nodes and parotids.

H. B. STALLARD.

(3) **Kruskal, I. D. and Levitt, J. M. (Brooklyn).—Uveoparotid tuberculosis.** *Amer. Jl. of Ophthal.*, Vol. XVIII, August, 1935.

(3) **Kruskal** and **Levitt** review the literature of uveo-parotitis and give a brief account of the main clinical features of this disease. They support the conception that tuberculosis is an aetiological factor.

They record 2 cases from their practices. One case, a married woman, 34 years of age, had an enlarged supra-clavicular gland removed and sectioned. The histological characters bore some resemblance to tuberculous lesions but showed no areas of caseation. Giant cells surrounded by endothelial cells and

lymphocytes are described but there is no note about the staining of these sections to show tubercle bacilli and none to the effect that a portion of the gland removed was minced and injected into guinea pigs. The authors state that in this case the Mantoux test was negative. Few pathologists would dogmatize about the histological appearances as shown in the excellent microphotographs which accompany this paper. It is evident that the cells are typical of those seen in chronic granulation tissue. In similar cases reported by other authorities the histology of the lesions has revealed no areas of caseation and tubercle bacilli have not been seen.

A slight anaemia, palpable spleen, intermittent pyrexia rising to 100·8°F. and bilateral enlargement of the lymph nodes at the hila of the lungs are other clinical features.

H. B. STALLARD.

(4) **Thiel, R. (Berlin).—Sunflower cataract in hepato-lenticular degeneration. (Sonnenblumenstar bei hepato-lentikularer Degeneration).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCIII, p. 12, 1934.

(4) **Thiel** records a case of hepato-lenticular degeneration (Kinnier Wilson disease) in a boy, 15 years of age. In addition to the neurological signs, involvement of the liver and the Kayser-Fleischer ring were present. The interest in this case was the presence of a sunflower cataract, typically seen in chalcosis, and previously noted in hepato-lenticular degeneration by Siemerling and Oloff and also by Vogt, who recorded his case in detail in the second edition of his Atlas. The possibility of the sunflower cataract in this affection being due to haematogenously deposited copper is discussed and its bearing on the nature of the pigmentation of the Kayser-Fleischer ring is discussed. A colour plate illustrating the appearances is given.

ARNOLD SORSBY.

(5) **Carroll, Frank D. (New York)—Analysis of fifty-five cases of tobacco-alcohol amblyopia.** *Arch. of Ophthal.*, September, 1935.

(5) **Carroll's** paper is based on the examination of 55 patients with tobacco-alcohol amblyopia. The condition would appear to be fairly common in New York, occurring in 0·3 to 0·5 per cent. of all new eye patients at one hospital. Among the 55 only two were women. The average age was 55·6 years, the youngest being 25 and the oldest 77. Onset was usually gradual. In one case which came for refraction 20/20 was obtained in each eye without a glass, and forty-nine days later the right eye could see only 10/200 and the left 20/100 with typical field changes. Five weeks

after giving up tobacco, vision had improved to 20/30. Illiterate patients complained surprisingly little about their symptoms while those who retained sufficient visual acuity to read found they could do so better with the left eye, than with the right, because of the position of the scotoma. This scotoma was caeco-central and usually much larger for red than for blue; in no case was there any contraction of peripheral fields to a 3/300 white object. Central vision was usually much diminished though one patient could see 20/20. No pathological changes were detected in the eyes except for the presence of pallor in the temporal halves of the discs in 19 patients and in 16, faint fine mottling of the macular area of the retina. With regard to aetiology the author states that it is often impossible to tell whether the amblyopia is due principally to the alcohol or to the tobacco. He records a case where cure followed abstinence from alcohol while cigarette smoking (14 packages a week) was continued. Susceptibility is of more importance than quantity, since some patients used only small amounts of alcohol and tobacco and yet developed amblyopia; none had diabetes but two showed a trace of glycosuria on one examination. None showed any hypersensitiveness to tobacco by intradermal tests. Abstention is the most important factor in treatment, but it is worthy of note that among 17 patients who did not give up their tobacco and alcohol, 3 improved.

F. A. W-N.

(6) **Böck, J. and Rizak, E. (Vienna).—Retinitis pigmentosa and gigantism. (Retinitis pigmentosa und Hochwuchs).** *Zeitschr. f. Augenheilk.*, Vol. LXXXIV, p. 48, 1934.

(6) **Böck** and **Rizak** recall the literature on the cerebral factor in the genesis of retinitis pigmentosa, and in reporting a case of this affection in a man, 28 years of age, the subject of gigantism of the late eunochoid type, they hold that the findings support the view as to the cerebral origin of the retinal degeneration. There was no history of heredity or consanguinity, but oliguria and diminished salt secretion were present. Therapeutic tests with pituitary extract, chloretone and harmin supported their reading of the case as one of affection or maldevelopment of the midbrain.

ARNOLD SORSBY.

(7) **Koyanagi, Y. (Sendai, Japan).—The behaviour of the retinal pigment epithelium in eclampsia. (Ueber das Verhalten des retinalen Pigmentepithelium bei Eklampsia).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCIII, p. 478, 1935.

(7) As opposed to the current views that eclamptic retinitis is caused either by the direct action of toxins on the retina or by a

vascular spasm, **Koyanagi** advances evidence that the changes are produced by the secretory activity of the pigment epithelium. From histological study of two cases he shows the presence of subretinal exudates made up of multiple drops which in part give the staining reactions of hyaline tissue. Moreover, the pigment epithelium shows in addition to its normal components bodies that he regards as the morphological precursors of the drop-like exudate. The intermediate stages in this transformation are traced and illustrated. The ultimate cause of these changes is traced to hyaline degeneration of the smaller choroidal arteries, and lecithin in the serum is perhaps the toxic agent responsible for the vascular changes.

ARNOLD SORSBY.

(8) **Much, V. and Hüppi, A. (Lucerne).—Acute optic neuritis with sudden transient blindness as the first sign in acute anterior poliomyelitis. (Akute Neuritis optici mit plötzlicher, vorübergehender Erblindung als Primärsymptom einer Poliomyelitis anterior acuta).** ***Klin. Monatsbl. f. Augenheilk.***, Vol. XCIII, p. 333, 1934.

(8) **Much** and **Hüppi** record the case of a woman, 37 years of age, who developed bilateral acute optic neuritis with complete blindness and inactive pupils. A week later typical symptoms and signs of acute poliomyelitis developed. Subsequently, some optic atrophy supervened but vision was practically fully restored, though a central scotoma and shrunken fields for colour persisted for some time; paresis of accommodation was also present for some time. The case is of interest in that the first sign of the widespread disease was ocular in nature; the circumscribed ophthalmoplegia interna and the optic neuritis must have been due to localized action of the virus. [The case is also of interest as a parallel to neuromyelitis optica.—A. S.]

ARNOLD SORSBY.

(9) **Brana, J. and Radnai, P. (Budapest).—Prognosis in hypertension on the basis of vascular changes in the fundus. (Die Prognose der Hypertonie auf Grund der Gefässveränderungen im Augenhintergrund).** ***Klin. Monatsbl. f. Augenheilk.***, Vol. XCIII, p. 455, 1934.

(9) In an article illustrated by a number of tables, **Brana** and **Radnai** give an analysis of 100 cases of vascular hypertension studied from both the general and ocular aspects. They find that the height of the systolic or diastolic pressure is no guide to prognosis in any individual case; much more can be learnt from the appearances in the fundus, even though the fundus changes

have no parallelism to the blood pressure. Classifying the fundus changes into three groups according to the degree of arteriovenous compression, they found that whilst in mild cases, the mortality within a year was 2 per cent., medium cases gave 60 per cent. and the severest, 100 per cent. (9 cases).

ARNOLD SORSBY.

(10) **Knapp, P. (Basle).—? Raynaud's disease. (Raynaudsche Krankheit?)** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCIII, p. 466, 1935.

(10) Knapp reports three cases suggestive of Raynaud's disease in patients, 38, 18 and 28 years of age. They all had phenomena of spasm of the retinal arteries and in one case, the onset and termination of the spasm was observed ophthalmoscopically. This attack only lasted one minute; the disc had become pale and the patient had the sensation of sudden clouding of vision. The author holds that the spasms are of nervous origin and that permanent narrowing of the arteries supervenes only if the attacks are persistent or of frequent recurrence.

ARNOLD SORSBY.

(11) **Damel, C. S. — Occlusion of the central artery of the retina. (Occlusion de la arteria central de la retina).** *Arch. de Oftal. Buenos Aires*, Vol. X, p. 395, June, 1935.

(11) Damel has studied in detail a case of occlusion of the central artery of the retina from 48 hours to 5 months after its occurrence. His article includes excellent photographs of the retina taken on the first visit of the patient, and showing the condition of the arteries, the disc and the macular region; photographs were taken at later intervals showing the re-establishment of the circulation and the changes taking place, especially in the macular region and at the disc. He discusses the time of onset of the macular oedema, and also the causes and time of appearance of the cherry red spot. The author quotes in some detail the opinion of various authorities on this subject, and he considers that the differences in appearance of the vessels in these cases of central block as described by other authors is simply due to the moment at which the patient was examined.

The author's patient had had a stroke 5 months before he suddenly lost the vision in the left eye, this stroke left him with a right hemiplegia.

The pathology of this condition has been treated in great detail.

E. E. CASS.

(12) **Terry, T. L. (Boston). — Angeoid streaks and osteitis deformans.** *Trans. Amer. Ophthal. Soc.*, Vol. XXXII, p. 555, 1934.

(12) **Terry** has investigated the incidence of angioid streaks in osteitis deformans. His paper describes the fundus appearances of angioid streaks and the histopathological findings of Verhoeff's case in which there was found localized proliferation of the retinal pigment epithelium in furrow-like creases, plication of Bruch's membrane and the choriocapillaris, fibrosis of the choroid, sclerosis of the choroidal vessels, tears and local separation of the choroid from the sclera and ruptures in Bruch's membrane.

In 106 cases of angioid streaks reported in the literature osteitis deformans was evident in 8·49 per cent., and all the cases in which these two diseases were associated exhibited marked bony changes in the skull. Angioid streaks may be present before osteitis deformas is clinically demonstrable. The author states that radiogrpahic evidence of osteitis deformans may be present before clinical signs appear and therefore urges the necessity for this investigation in suspected cases. He believes that arteriolar sclerosis combined with some other pathological factor, at present unknown, may initiate changes in bone and in the choroid producing sclerosis in these structures.

Of 22 cases of osteitis deformans examined by the author 3 showed angioid streaks, 4 a 'halo' around the optic disc due to loss of pigment and degeneration in the choroid, one choroidal fibrosis, 3 colloidal excrescences in the fundi, 5 retinal arteriosclerosis, 2 retinal haemorrhage, 3 choroidal haemorrhage, 1 central retinal vein thrombosis and 1 case showed pseudoxanthoma elasticum.

Walker has pointed out the similarity between the structure of the choroid and that of the organ of Corti and suggests that sclerosis in these structures might explain both the angioid streaks and deafness associated with osteitis deformans.

H. B. STALLARD.

(13) **Pavia, J. L. (Buenos Aires).—Papilloedema and varicella. (Edema de papila y varicela).** *Rev. Oto.-Neuro. Oftal.*, Vol. IX, p. 1, 1935.

(13) **Pavia** states that only two cases have been recorded in which alterations in the optic nerve have occurred during an attack of chicken-pox.

The first is a case recorded by Hutchinson in the *Ophthalmic Review* of September 1886. The other is recorded by Chavernac in 1906.

The author's case is one of a child, 9 years of age, who had had chicken-pox a month before; the temperature had been high for 5 days, the child had complained of pain in the limbs, it frequently vomited and had severe headaches. After the fever had left him for 15 days, he went back to school, and it was then found that he could not see to read nor write.

On examination, the right vision was 5/10 and the left vision was 6/10. There was a papilloedema in both eyes and also an oedema of the surrounding retina.

X-rays of the skull showed nothing abnormal. The Wassermann reaction was negative, the cerebro-spinal fluid pressure was "40" and came down to "10" after 8 c.c. had been extracted. Nothing abnormal was found in it, but immediately after the lumbar puncture, the child felt better. He was given 10 injections of 25 per cent. glucose intravenously. At the end of a month the right vision had improved to 8/10 and the left to 9/10. There was then some pallor of the discs, which were of normal size and had well-defined edges. The veins and arteries were normal in calibre, but in the macular region there still remained some slight oedema.

The author gives the details of Chavernac's case in which there was also a rapid improvement in the vision.

Pavia suggests that the oedema in these cases is part of the picture of a very mild serous-meningitis, and says that it very frequently follows infectious conditions in children. He himself has observed it in cases of pneumonia, influenza, erysipelas and typhus, and above all in cases following infections of otic origin.

He draws attention to the fact that excellent results are obtained from lumbar puncture and hypertonic injections, both of which are active agents in bringing down intra-cranial pressure.

E. E. CASS.

II—MEDICAL OPHTHALMOLOGY

(1) **Steen, R. E. (Dublin).—Tay-Sachs' disease. (Amaurotic family idiocy).** *Irish Jl. of Med. Science,* September, 1935.

(1) **Steen** prefaces the account of his case with an excellent review of the disease. His patient was a girl, aged one year and 10 months, admitted to Dr. Steevens's Hospital in March, 1933.

The child had thrived up to the age of nine months, when her weight was 23lbs. She was holding her head up and using her arms and legs, and though she had not been able to sit up of her

own accord, she was able to reach out for things and to recognise her parents.

At nine months she became quiet, and later did not seem to recognise people. Her weight, at just under the age of one year, was nearly 32lbs., but it dropped steadily until, on admission, it was 18lbs. 4oz.

The family history is of much interest, no Jewish blood could be traced on either side of the family. In 1920-21 the father had been in a mental hospital suffering from delusion. He had apparently quite recovered and was at work. There were 11 children, of whom the patient was the youngest. The eldest, a boy, aged 20 years, is perfectly healthy. Twins followed, one, a boy, died at the age of five months from convulsions; the other, a girl, died from the effects of burns, at the age of nine years. The fourth member of the family is a healthy boy, aged 17 years. The fifth, a girl, aged 14 years 6 months, is healthy. Next came twins, prematurely; one stillborn, the other surviving 24 hours. The eighth member is a girl, aged 10 years, and healthy. The ninth, a boy, would seem to have been a case of Tay-Sachs' Disease. He did well until nine months of age, then deterioration appeared, and he died at the age of 21 months. The mother said that the course of the illness was exactly similar to that of the youngest child. The tenth member of the family is a girl, aged 4 years, and healthy.

Steen's patient on admission lay on her side and took no interest in her surroundings. The fontanelle was open and the head circumference was 19·75in. The teeth were backward; an almost constant chewing movement of the mouth was noted, with occasional facial twitching, like that seen in uraemia. Chvostek's sign was negative. Feeding was badly performed; no retraction of the head was present, no pain or resistance on flexing the head; and marked wasting was evident everywhere. Heart, lungs, abdomen and urine were normal.

The attitude was in some respects like that of decerebrate rigidity. The legs were extended, the feet, plantar-flexed, the arms flexed slightly at the elbows, markedly flexed at the wrists and finger joints, with inversion of the thumbs.

The pupils were equal, rather large, and reacted sluggishly to light. Well marked nystagmus was present, strabismus was noticed occasionally. Hearing was hyperacute. General wasting of the muscles was apparent, the deep reflexes were all exaggerated. Incontience of urine was present throughout her stay in hospital. The fundus showed the typical picture on each side.

The article is illustrated by excellent pictures showing the child with typical attitude of the limbs and characteristic position of the

hand and wrist. There is also a monochrome plate of the fundi. The age of the child at death is not given.

R. R. J.

(2) **Bhaduri, B. N. (Calcutta).—Papilloedema in epidemic dropsy.** *Calcutta Med. Jl.*, August, 1935.

(2) **Bhaduri** read a paper on this subject at the Fourth Annual Conference of the All-India Ophthalmological Society in April, 1935. He reported nine cases of papilloedema in epidemic dropsy. In his present article a further case is reported, and the suggestion is advanced that the swelling of the nerve-head is probably due to raised intra-cranial pressure resulting from excess formation of cerebro-spinal fluid due to the involvement of the choroid plexuses in epidemic dropsy.

His case was that of a Hindu male, aged 43 years. He had suffered from epidemic dropsy for two months.

The intra-ocular tension was raised to 40 mm. Schiötz in the right eye and 32 mm. in the left, central vision was good. Examination of the field showed a full periphery and slight increase in the size of the blind spot.

There was 4 to 4·5 D. swelling of the right disc and 5·5 to 6 D. on the left side. The left fundus, in addition, showed massive haemorrhages on the disc and in the surrounding retina. The Wassermann reaction in the blood was negative and the urine normal. The patient refused to submit to lumbar puncture A month later the patient was much better and he then ceased attending. Complete subsidence of the papilloedema in this disease usually occurs within two to three months.

An excellent coloured plate of the fundus accompanies the paper.

R. R. J.

(3) **Simões, F. (Bahia, Brazil).—A case of unilateral papilloedema. (Um caso nevite optica edemotosa unilateral).** *Rev. d'Ophtal. de Sao Paulo*, Vol. CXI, p. 244, 1934.

(3) **Simões** describes a case that came to the Santa Isabel Hospital complaining that the vision in the right eye had been a little diminished for a few days. The patient admitted that he had had syphilis. He was diagnosed as optic neuritis and was treated with injections of 914. Twelve days later he developed an intolerable headache and on examination was found to have a marked oedema of the right optic disc. The headache became more and more severe, and a lumbar puncture was performed for diagnostic purposes. There was a marked increase of pressure of the cerebro-spinal fluid, which contained 0·6 per cent. albumin and 986 cells per cubic centimetre.

The theories of causation of a unilateral oedema are discussed at length. The author thinks the reason in this case was compression of the optic nerve with inflammatory oedema. He gives the differential diagnosis from cerebral tumour and other lesions. The patient was given urotropin and bismuth intravenously and another lumbar puncture was performed 15 days later. After this the oedema of the disc began to subside and finally the fundus returned to normal.

The author then discusses the action of arsenic on the optic nerve and also the "pros" and "cons" of giving arsenic in syphilitic cases where the optic nerve is affected. He discusses the relative toxicity of the various arsenical products used as anti-syphilitic remedies.

E. E. CASS.

(4) **Bhaduri, B. N. (Calcutta).—Ocular complications in cholera.** *Calcutta Med. Jl.*, October, 1935.

(4) Bengal is the home of cholera. Ocular changes in the disease have been noted since 1846, but since the appearance of Elliot's "Tropical Ophthalmology" few papers have been published.

Bhaduri has had experience of cholera cases in the Carmichael Medical College Hospital and in private practice.

After a brief discussion of the various ocular symptoms observed by others the author states his own findings.

"The pupillary reaction cannot be treated as of any prognostic importance regarding life." He has also observed that owing to the damaged epithelium of the cornea, due to the enormous loss of fluid in this disease, very weak hypo- or isotonic solutions of mydriatics give a maximum and prolonged mydriasis.

Lens opacities were seen in only two cases, and they were cases of senile cataract in patients who had contracted cholera. In a third patient, aged 19 years, nuclear sclerosis was found in one eye. It persisted after recovery.

No striking changes were noted in the papilla in his series, but an optic atrophy case became quite blind on the fourth day after developing cholera.

Dark discolouration of the retinal veins was very noticeable; not so in the arteries. A broken column in the veins was noted in three cases.

The intra-ocular tension was, of course, lowered. The author finds that the intra-ocular tension varies almost inversely with the specific gravity of the blood in cholera cases.

R. R. J.

(5) **Granström, K. O. (Stockholm).—Changes of refraction in diabetes. (Refraktionsveränderungen bei Diabetes Mellitus).** *Acta Ophthal.*, Vol. XI, p. 1, 1933.

(5) **Granström** gives an exhaustive survey covering 160 pages on the changes in refraction in diabetes. There is a detailed study of 77 cases in addition to a review of the literature. He found that generally the changes in refraction are in the direction of an increase of the total refraction of the eye. Decrease in refraction occurs only in untreated cases of diabetes; such decrease ceases as soon as excretion of sugar is controlled therapeutically. He also points out that it would appear that increase of refraction can also occur if excretion of sugar is diminished for other than therapeutic reasons. Changes of refraction occur frequently at a time when diabetics are first treated, irrespective of whether insulin is used or not. In the later stages in the treatment of diabetics, increase of refraction is less frequent. Changes in refraction seem to be distributed equally over different ages and they are not more common at the presbyopic age, though marked changes in refraction seem to be definitely more frequent in young individuals. A transient increase in refraction occurs a few days after the beginning of treatment and generally lasts from two to four weeks. Regression is slower than the development of these transient refractive changes and in half the cases was not associated with any subjective symptoms. The author could find no association between the presence of acetone bodies and acidosis and changes in refraction, nor could he establish any relationship between the state of partial hunger during treatment and decrease of refraction. As regards the cornea, the author confirms the observation of earlier investigators that no changes in the radius of curvature occur. He further found that there is no change in the position of the macula. Increase in refraction was associated with an increased positive aberration, decrease with a diminished positive aberration. The changes in refraction seemed to be localised to the axial part of the refractive media. Transitory changes in refraction were found to be frequently associated with a diminished range of accommodation in patients under the age of 40. This was practically the rule in patients under the age of 30. After 30 there seems to be no effect on a range of accommodation. The changes in refraction are caused by changes in the lens. Stigmatoscopic investigations tend to show that these changes are intracapsular and are probably caused by changes in the index power in the central parts of the lens, a view which is supported by the findings as regards accommodation. He concludes by discussing the possibility of the changes in the lenses being caused by a disturbance in the water and salt metabolism in diabetics.

ARNOLD SORSBY.

(6) **Villani (Rome).—The lesions of the pigmented epithelium of the iris and ciliary body in artificial diabetes. (Studio istologico delle lesioni dell epitelio pigmentato dell'iride e dell'epitelio del corpo ciliare del diabete sperimentale).** ***Ann. di Ottal.***, September, 1934.

(6) It is well known that the pigment epithelium of the iris is altered and the pigment loosened in glycosuric patients. Villani has produced glycosuria artificially in rabbits and has examined the pigment epithelium; he finds that degeneration is constant, under one of two forms: either there is granular degeneration of the cell-protoplasm with alteration of the chromatin of the nucleus, or there is vacuolated degeneration of both parts of the cell. At a late stage there is necrosis of the cells and a complete loss of all the pigmented epithelium from the back of the iris.

HAROLD GRIMSDALE.

III.—MISCELLANEOUS

(1) **Cavallacci (Pisa).—The effect of irradiation with ultra-violet rays on the ascorbic acid content of the rabbit's eye. (Sul comportamento dell acido ascorbico (Vitamin C) nell'occhio di coniglio irradiato con raggi iltravioletti).** ***Arch. di Ottal.***, May-June, 1935.

(1) **Cavallacci** discusses first the relation of ascorbic acid to vitamin C. He concludes that it is probable that vitamin C is not a simple body but that there are other constituents in addition to ascorbic acid; this, however, has the same property of protecting against scurvy, though according to some observers it is not so powerful.

Experiments have shown that this acid is present in considerable quantity in the lens and in the aqueous. In the aqueous it is much more abundant than in the blood and it has been suggested that it is manufactured in the lens and thence comes into the aqueous. Further knowledge on this point is very desirable.

In the experiments on which this paper is based, the author has subjected one eye of rabbits to irradiation by ultra-violet rays for various periods and has compared the content of ascorbic acid with that of the other eye. He supposed that the increased chemical activity under the rays would alter the amount of the substance; he finds a reduction in all the tissues of the eye. He thinks that in the retina the reduction is due to increased oxidation accelerated by the action of the rays; but that in the lens and humours, it is secondary to the intense congestion which follows exposure to ultra-violet light.

HAROLD GRIMSDALE.

(2) **Pierguidi (Trieste).—The glutathione content of the ocular liquids and tissues. (Sul contenuto in glutatione di alcuni liquidi e tessuti oculari).** *Boll. d'Ocul.*, July, 1935.

(2) A very important factor of internal respiration is glutathione, a compound of two molecules of cystein; these combining give up H_2 acting as reducing agents, and become cystine; under certain circumstances this takes up the H again and acts as an oxidiser. These reactions are constantly reversible.

Pierguidi finds that the cornea contains a large amount of glutathione. It was to be expected that the avascular cornea would hold in its tissues a large amount of substance to aid in its internal respiration. This is even more important for the lens which has to rely entirely on such substances to supply the oxygen necessary for its metabolism; it would seem that the lens has the power of selecting glutathione from the ocular liquids and storing it in quantity.

HAROLD GRIMSDALE.

(3) **Villani (Rome).—The healing of wounds of the lids. (Studio sperimentale sulla riparazione delle ferite delle palpebre).** *Boll. d'Ocul.*, August, 1934.

(3) Little is known of the process of healing in wounds of the lids. Villani has attempted to fill this gap in our knowledge by experiments on rabbits. They show that wounds that lie transversely in the lid heal rapidly, since there is no tendency for the edges to become separated. Vertical wounds, involving the free margin of the lid are much more likely to be followed by deformity, since the edges are drawn apart by the orbicularis. To secure rapid healing without deformity it is advisable to suture the conjunctiva since this may become ntro- or extroflexed, and it is especially necessary to secure accurate coaptation of the two parts of the free margin.

The author notes that in his experiments, the Meibomian glands in most cases undergo necrosis in whole or part.

HAROLD GRIMSDALE.

(4) **Anelli (Rome).—The permeability of the lacrimal passages in dacryocystitis. (La permeabilità delle vie lagrimali nelle dacriocistiti).** *Boll. d'Ocul.*, August, 1934.

(4) It is generally assumed that dacryocystitis follows on stricture of the nasal duct. Ovio thinks that stricture, on the other hand, follows dacryocystitis. Anelli has examined the condition of the passages in 100 cases. He has tested the freedom of the passage by instilling a coloured solution into the conjunctival sac

and observing the possible colouration of a pad of cotton wool in the nostril of the same side. If the passage is pervious, the colour will be seen within a few minutes. He finds that in the early stage of dacryocystitis, when the contents of the sac are not purulent, the passage is sometimes free, but when the process is more advanced, the passages are always completely obstructed. He thinks that generally the inflammation of the duct which leads to obstruction, has origin in inflammation of the nasal mucous membrane, which spreads to the lining of the duct.

HAROLD GRIMSDALE.

(5) **Reed, H. and Meyer, L. (Chicago).—Bilateral temporal pterygia.** *Amer. Jl. of Ophthal.*, Vol. XVIII, July, 1935.

(5) **Reed** and **Mayer** report a case of a Chinese male, 49 years of age, in whom bilateral temporal pterygia were present and the conjunctiva on the nasal part of the globe was unaffected by this disease. They comment that this is the only case reported in the literature up to date in which these clinical features are present.

H. B. STALLARD.

(6) **Kraupa, E. (Brünn).—Late glaucoma in interstitial keratitis. (Das Spätglaukom nach Keratitis parenchymatosa).** *Zeitschr. f. Augenheilk.*, Vol. LXXXIV, p. 43, 1934.

(6) **Kraupa** reports three cases of late secondary glaucoma in interstitial keratitis, in patients aged 36, 52 and 56. In the first case cyclolialysis was needed after iridectomy; in the second cyclodialysis after trephining. In the third case an iridectomy by itself was successful. He argues that the prognosis is not so bad as is generally believed, especially if cyclodialysis is performed, but this operation is advised as the first interference only when the iris is heavily damaged by the inflammation.

ARNOLD SORSBY.

(7) **Cepero, G. and Gomas, L. (Havana).—Treatment of iritis by diathermy. (Die Diathermie Behandlung der Iritis).** *Zeitschr. f. Augenheilk.*, Vol. LXXXIV, p. 211, 1934.

(7) **Cepero** and **Comas** relate their experience of the use of diathermy in 230 cases during the course of two and a half years. They speak enthusiastically of its value in iritis, having experience of 57 cases. They use a frequency current of 2 million and hold that the effect is produced not merely by the heat engendered but by protoplasmic changes. As contra-indications they give haemorrhagic processes as also intra-ocular septic lesions. An

average of 7 to 10 sittings are necessary; atropine is the one other treatment employed and its effect is much enhanced by the diathermy.

ARNOLD SORSBY.

(8) **Cogan and Cogan (Boston).—Dinitrophenol cataract.** *Jl. Amer. Med. Assoc.*, September 7, 1935.

Lazar (Chicago).—Cataract following the use of dinitrophenol. *Jl. Amer. Med. Assoc.*, September 7, 1935.

Kniskern (Michigan).—Cataracts following dinitrophenol. *Jl. Amer. Med. Assoc.*, September 7, 1935.

Allen and Benson (Chicago).—Late development of cataract following use of dinitrophenol about a year before. *Jl. Amer. Med. Assoc.*, September 7, 1935.

(8) The dangers of slimming forms the topic of a leader in the *Jl. of the Amer. Med. Assoc.* of the above date. "It is interesting to note that all the so-called reducing preparations on the market fall into three categories: first, laxatives that deny the body the benefit of its food intake, as the salts, crystals and herb teas; second, obvious frauds that depend for effect upon the stringent diets prescribed as part of the treatment; and third, the unquestionably effective but dangerous articles containing thyroid or dinitrophenol, both of which act by speeding up the utilization of food. All of them are unwarranted impositions on the public, which cannot evaluate claims made for the preparations and cannot readily appreciate the harm that may result from careless use of the products."

The cases reported are those of comparatively young individuals who took dinitrophenol for the purpose of relieving obesity. The treatment was efficacious but in each case was accompanied by rapid opacification of the lenses, mainly in the posterior cortex.

A. F. MACCALLAN.

(9) **Sala (Palermo).—Chalcosis (staining by copper) of the eye with special regard to copper cataract. (Sulla calcosi oculaire con particolare riguardo all'aspetto biomicroscopico della cataratta da rame).** *Boll. d'Ocul.*, August, 1934.

(9) It has been recognized for a long time that a copper foreign body is able to set up violent inflammatory changes in the eye, even though it may have been introduced aseptically. The characteristic colouration of the various structures has been noted comparatively recently, and the name chalcosis, corresponding to siderosis is a recent coining.

Cataract from copper presents certain features; it is generally discoid in shape of the size of a normal pupil with rays and is

compared to a sunflower; it is greenish in colour and is invisible to transillumination; it is situated in the anterior part of the lens, in the anterior capsule or the adjacent epithelium. The cause of the shape has been supposed to be the pressure of the iris, but **Sala** thinks that this cannot be the sole cause as in his case the cataract was completely within the area of the normal pupil.

HAROLD GRIMSDALE.

(10) **Camozzi (Como).—The assessment of indemnity for injuries of the eyes. (Sulla valutazione del danno nella infortunistica oculaire).** *Lett. Oftal.*, January, 1935.

(10) The question of compensation for the partial loss of vision from an accident is a difficult one. Many have tried to formulate standards which shall apply to all the variations and have not succeeded; it has not even been possible to find agreement about the total loss of one eye.

The congress at Rome in 1920 held that 7/10 of normal vision was all that was necessary for carrying on most work, and therefore concluded that any loss not exceeding this, deserved no compensation. But two points have since been agreed upon with practical unanimity. (1) That it is absurd to reckon compensation arithmetically as fractional loss of acuity without taking into consideration the collateral visual functions; (2) that compensation must be relatively greater, the greater the loss of vision, since the small loss entails no disability.

The capacity for work depends on many factors as well as the visual; and thus varies with the individual. Age has an important bearing. Is it just, asks **Camozzi**, to give the same amount of compensation for an equal loss of sight to a young man in the prime of life for whom the future may hold great possibilities, and to an old man for whom the future holds no prospect? Or is it right to base compensation in all cases on the wages received at the moment of injury, and thus to exclude the possibility of advancement?

The author suggests that there should be a body of impartial experts to examine and adjudge all cases of compensation, with a Court of Appeal for those not satisfied by their findings.

HAROLD GRIMSDALE.

(11) **Bellows, John C. (Chicago).—Surface anaesthesia in ophthalmology.** *Arch. of Ophthal.*, December, 1934.

(11) **Bellows'** experiments were performed on human corneae and the depth of anaesthesia was tested with von Frey's hairs. One drop of each solution was used, and the order of their

effectiveness was 0·5 per cent. p-n butylaminobenzoyl-dimethylanimo-ethanol hydrochloride; 1 : 500 nupercaine, 1 per cent. butyn, 2 per cent. cocaine, 1 per cent. phenacaine and 2 per cent. metacaine. The effectiveness was measured in units which represented the average depth of anaesthesia multiplied by its duration in minutes. Except with nupercaine there was insensibility to the initial stimulus within 30 seconds and the anaesthaesia was at its maximum in 2 minutes; with nupercaine, the figures were 45 seconds and 5 minutes respectively. Cocaine was the only drug in the series that produced drying of the corneal epithelium and enlargement of the pupil and palpebral fissure. Butyn caused the least and phenacaine the greatest pain and congestion.

F. A. W-N.

(12) Neuschüler, Ignagio (Rome).—Complete bilateral external ophthalmoplegia from spinal anaesthesia. (Oftalmoplegia esterna completa bilaterale da rachianestesia). *Rev. Oto-Neuro.-Oftal. E Radio.-Neuro.-Chirur.*, Vol. XI, No. 3, p. 255, May-June, 1934.

(12) Since the improvement in technique and the adoption of anaesthetics other than cocaine, the accidents following spinal anaesthesia have diminished considerably. Complications may follow a simple lumbar puncture; after a lumbar puncture there is always a fall of pressure followed by a prolonged rise.

Dr. Neuschüler mentions a case of complete external bilateral ophthalmoplegia in a boy, 16 years of age, following a spinal anaesthetic, on examination, however, he was found to have some slight hyperthyroidism and a latent disease of the nervous system, which was exacerbated by the anaesthetic. Other cases have been recorded of paresis of eye muscles (following a lumbar puncture) in which there was latent cerebral syphilis. In the case the author himself saw and described, there was no pre-existing nervous or organic disease. There was an external ophthalmoplegia of gradual onset, starting 4-5 days after operation and complete in both eyes in 7-8 days. This started to clear at the end of a week and only paresis of both 6th nerves remained after 4 months. Recovery was complete at the end of the year. There were never any signs of increased intra-cranial pressure.

Immediate manifestations after lumbar puncture are due to affection of the 4th ventricle or to bulbar anaemia.

Position of the patient during operation is of importance. Most eye complications follow the Trendelenburg position. Various theories of formation and circulation of the cerebro-spinal fluid are described. The author suggests that the causes of the complications following spinal anaesthesia may be:—

1. Hypertension due to serous meningitis. (Inhibition of absorption of the cerebro-spinal fluid or increased formation).
2. Hypotension.
3. Basal meningitis without effusion, which may cause pressure on the nerves.
4. A neuritis due to a chemical union of the alkaloid with the nerve cells.
5. Exacerbation of a previous organic (*i.e.*, endocrine or nervous) lesion.
6. Toxicity due to chemical alterations in the anaesthetic.

E. E. CASS.

BOOK NOTICE

L'Extraction Capsulo-Lenticulaire de la Cataracte. By R. DE SAINT-MARTIN. 483 pp., 76 illustrations, and 15 coloured drawings. Paris: Masson et Cie. 1935. 120 francs.

This is a most elaborate book, published under the auspices of the *Soc. franç. d'Ophtal.*, and forms one of the collection which includes the several monographs on slit-lamp microscopy and Dr. Redslob's work on the vitreous body.

It is the largest book we are acquainted with treating of intra-capsular extraction of the lens.

In the first chapter the author gives his reason for choosing the title of the book, and why he has discarded "Total extraction," "Intra-capsular extraction," and "Extractions *in toto*." He points out that the title he has chosen includes not only those cases in which the lens is removed in the complete capsule, but those in which the lens and the capsule are removed.

There follows a chapter on the historical aspect, and it is shown that in 1751 Pellier de Quengsy Père removed a cataract in its capsule by means of forceps, and Samuel Sharp in 1753 described cases of the intra-capsular operation that he had performed in that year.

Intra-capsular extractions may be classified under three headings:—

(1) Technique by pressure alone as done by Colonel H. Smith;
(2) Technique by traction:
  (*a*) with forceps;
  (*b*) with a pneumatic sucker;
(3) Technique by a combination of pressure and traction.

There follows a chapter showing the anatomical possibility of the operation together with an elaborate consideration of the parts involved. This concludes the first part of the book.

The next part deals with the general examination of the patient such as we are quite familiar with, and then an important chapter on the preparation of the patient. From this chapter much may be learned, not only by those who are advocates of the intra-capsular operation, but also by those who practise the operation preceded by capsulotomy.

Four, five or even 10 per cent. solution of cocaine hydrochloride is used for anaesthesia combined with 1/1,000 adrenalin solution. The author prefers adrenalin mydriasis produced by injecting two drops of 1/1,000 adrenalin solution at the limbus below.

Then follows a description of the methods of paralysing the eyelids, together with a retro-bulbar injection of novocaine and adrenalin. All these preparations are carried out an hour or so before the operation; at the time of operation the superior rectus is paralysed by an injection of novocaine.

A suture through the superior rectus tendon is always applied.

Up to this point there is no detail that may not be used with advantage in any method of cataract extraction. Without doubt the introduction of paralysis of the orbicularis palpebrarum has been a help of the first importance in cataract extraction, the first important improvement since the introduction of extraction of cataract through an intact pupil, thus abolishing the bugbear of entanglement of capsule in the wound.

A retro-bulbar injection of novocaine and adrenalin is necessary in intra-capsular extractions, and when properly applied it has not led to haemorrhage into the orbit. There is reason to believe that it is of advantage in extraction following capsulotomy, not only by its anaesthetic effect, but also by the lowering of intra-ocular pressure, thus minimising the risk of loss of vitreous.

A suture in the tendon of the superior rectus is of advantage in all forms of cataract extraction.

It would seem that intra-capsular extraction is usually accompanied by some method of closing the wound, either by a suture or a conjunctival flap. Dr. Saint Martin advocates a suture applied before the section is made. This suture is most ingenious as, not only may the wound be rapidly closed, but the corneal flap may be manipulated by this means. The incision must include 180° of the cornea, followed by a small peripheral iridectomy.

The lens is then removed either by forceps or a suction apparatus accompanied by pressure over the limbus below. In this manner the lens is always "tumbled." It should be noted, however, that other operators prefer to deliver the upper pole of the lens first, in which circumstance a complete iridectomy must be performed.

Minute details are given concerning the tying of sutures and the toilette and reduction of the iris.

Under the head of dressing of the wound we are introduced to a

variety of shells, including those designed by the author and others.

The description of the operative technique of removal of the lens in its capsule is most minute and clear, and there are long discussions as to the advantages of the various means of delivering the lens, the author advocating either forceps or pneumatic sucker, discarding removal by pressure.

Chapters follow that deal with the subsequent course of the case, complications both at the time of operation and afterwards, most of which are familiar to ophthalmic surgeons whatever may be their method of operating.

There are many tables of statistics showing the relative frequency of these complications. A chapter is devoted to the slit-lamp examination of the operated eye with many coloured drawings.

There are interesting observations on the lens itself after removal and the deformities that are produced by the instrument with which the lens has been seized.

The last chapter deals with results, and there seems to be no doubt that in skilful and experienced hands the results following removal of cataract by traction, when serious attention has been paid to all points of technique, are beautiful. Loss of vitreous is certainly more common than in extraction after capsulotomy, but there are none of the complications associated with capsule and lens remains. The eyes are singularly free from iritis, and frequently mydriatics are not used from beginning to end. Provided every detail is followed minutely, then, in skilful hands intra-capsular extraction has much to commend it, but if intra-capsular extraction is undertaken making use only of the older methods of anaesthetisation, the usual types of lids speculum, and so on, then the operation is extremely dangerous, and anyone who would devote himself to intra-capsular extraction as a routine operation, should watch the operation being performed by experts, since small errors in technique may wreck an eye.

Those of us who still practice extraction through an intact pupil after capsulotomy or the use of capsule forceps, followed by lavage of soft matter and peripheral iridectomy followed in four to six weeks by a gently performed and well-planned capsulotomy will probably feel that this technique has not been sufficiently considered by advocates of intra-capsular extraction who stress so much the dangers of post-extraction capsulotomy.

On the other hand, those who do not practice or agree with intra-capsular extraction should read this book carefully, not only for its absorbing interest, but because they will see weak points in their own technique and find so many means whereby their own operating may be improved.

NOTES

Moorfields Reports

AS all know the Moorfields Reports began in 1857 and the last volume was published in 1917 just before the amalgamation of the three eye periodicals in the *British Journal of Ophthalmology*. Complete sets of the Reports are valuable and difficult to obtain.

At the present time the journal holds in store a large number of back volumes and spare parts.

The management is prepared to consider the sale of such volumes or parts to ophthalmic surgeons who wish to make up as complete a set as is possible in existing circumstances. The price per volume has been fixed at half-a-guinea; and 3/6 per single part. Carriage abroad will be extra.

Tenders and enquiries should be addressed to the Manager.*

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Appointments

Mr. A. C. HUDSON has been elected consulting ophthalmic surgeon to St. Thomas's Hospital. Mr. P. G. Doyne has been appointed ophthalmic surgeon and Mr. G. G. Penman has been appointed ophthalmic surgeon-in-charge of out-patients at St. Thomas's.

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Ophthalmological Society of the United Kingdom

THE Annual Congress will be held in London from April 2nd to 4th, 1936. The subject for discussion is "Ophthalmia Neonatorum," to be opened by Mr. J. G. Magor Cardell, Dr. S. H. Browning and an obstetric surgeon. On the afternon of April 3rd, there will be five-minute demonstrations of exhibits of scientific material and apparatus. The Annual Dinner will be held on April 2nd.

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Corrigendum

IN the account of the Twenty-first Anniversary Meeting of the North of England Ophthalmological Society (Vol. XIX, p. 678), we were in error in stating that the presentation to Mr. Percival Hay was made by the North of England Society as an official gift from the Society. The presentation was made on behalf of those who took part in the ophthalmological Continental tour last year.

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Royal London Ophthalmic (Moorfields) Hospital, Annual Dinner

THE Annual Dinner of past and present students of the Royal London Ophthalmic Hospital will take place at the Langham Hotel, Portland Place, W.1, on Thursday, March 12, 1936, at 7 for 7.30 p.m.

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* Volumes I and II are unobtainable; Vols. III, VI, VII, VIII, IX, X, XIII and XIV cannot be supplied at the above prices.

The chairman will be Sir John Herbert Parsons, D.Sc., F.R.C.S., F.R.S., consulting surgeon to the Hospital.

As the meeting of the ophthalmological section of the Royal Society of Medicine takes place at the Hospital on Friday, March 13, there will be an opportunity for old students who come to the dinner to see the new buildings that have been opened during the past year.

Those who wish to attend the Dinner should send their names to Mr. P. G. Doyne, 60, Queen Anne Street, W.1, enclosing a cheque for 15/-, which is the price of the ticket (excluding wine).

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Death As we go to press we learn with much regret that Mr. Frank Cowley, of Dublin, died suddenly on December 15.

FUTURE ARRANGEMENTS

1936

January 10.—Royal Society of Medicine, Section of Ophthalmology.

February 4.—Midland Ophthalmological Society, at Stoke (Royal Infirmary).

February 7.—North of England Ophthalmological Society, at Newcastle-on-Tyne.

February 14.—Royal Society of Medicine, Section of Ophthalmology.

March 6.—North of England Ophthalmological Society, at Liverpool.

March 12.—Royal London Ophthalmic Hospital, Annual Dinner.

March 13.—Royal Society of Medicine, Section of Ophthalmology (Clinical Meeting).

March 27.—North of England Ophthalmological Society, at Sheffield.

April 2-4.—Ophthalmological Society of the United Kingdom (Annual Congress), in London.

April 3.—Midland Ophthalmological Society, at Nottingham Eye Infirmary.

May 22.—Midland Ophthalmological Society, at Addenbroke's Hospital, Cambridge.

June 12.—Royal Society of Medicine, Section of Ophthalmology (Annual Meeting).

October 6.—Midland Ophthalmological Society, at Birmingham Eye Hospital.

December 1.—Midland Ophthalmological Society, at Birmingham Eye Hospital.

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *November, 1935.*

LAMBERT. Studies of the retinal circulation by direct microscopy.
AMES. Aniseikonia—a factor in the functioning of vision.
MCKEE. A study of the pneumococcus group from the inflamed conjunctiva and lacrymal sac.
DAVIDSON. The traumatic ophthalmoplegias as a workmen's-compensation problem.
TINKER. Illumination intensities for reading.
MANN. Newer developments in photography of the eye.
VON DER HEYDT. Ocular chalcosis.
BOSHES and MAYER. The lid-closure reflex of the pupil.

Archives d'Ophtalmologie. *November, 1935.*

OPIN and REBOUL. Marginal corneal dystrophy in young subjects.
VEIL and DOLLFUS. Comparative value of the different techniques for the obliteration of retinal tears.
RUBBRECHT. Reflections on the pathogenesis of retinal detachment.
BISTIS. The allergy of the eye.
DIMISSIANOS. Xanthomatous form of tuberculosis of the conjunctiva.
PERRIN and NOEL. A case of retinal neuro-epithelioma.

Annales d'Oculistique. *November, 1935.*

ANDRADE. Bilateral epithelioma of the corneo-scleral margin.
DEHOGUES. Exophthalmos from an orbital tumour.
AVERBACH and IVANOVA. Twelve hundred dacryocystorhinostomy plastic operations.
VIANNA. Auto-examination of the fundus.

Annali di Ottalmologia e Clinica Oculistica. *September, 1935.*

TROVATI. The physico-chemical constancy of the blood in glaucoma.
MARCHESINI and GHIO. Radiographic investigation of the orbital pyramid by means of the introduction of X opaque substance. (Experimental research.)
BUCALOSSI. Sarcoma of the optic disc. (Clinical and histological contribution to the study of tumours of the optic disc.)
MARCHESINI. The behaviour of the scleral canals and their relations with the venae vorticosae at various ages.
POSSENTI and CASTRIGNANI. Aetiology and pathological anatomy of pterygium.

Archivos de Oftalmologia Hispano-Americanos. *November, 1935.*

MÁRQUEZ. A new scheme of action of the ocular muscles for the better understanding of a frequent error in the interpretation of diplopia.
MOREU. New considerations on the pathogenesis, diagnosis and treatment of tuberculous infiltration of the cornea. (Parenchymatous keratitis.)
LÓPEZ. Some aspects of dental focal infection in ophthalmology.

Revista Oto-Neuro-Oftalmologia. *October, 1935.*

PAVIA and DUSSELDORP. Retinal detachment. Simultaneous scleral transillumination and diathermy.
HERNÁNDEZ, PAVIA and DUSSELDORP. Bacillaemic tuberculosis and scleral tuberculoma.

Revista de Ophthalmologica. *October, 1935.*

ALVARO. Classification of the causes of blindness.
FALÇÃO. Ocular tuberculosis and tuberculin.